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CLINIC OF DR. ARTHUR R. ELLIOTT

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## ABDOMINAL REFLEX DISORDERS

**Influence Exerted by Disturbing Emotional States on the Autonomic Nervous System and the Secondary Effects Thereof. Frequency of Abdominal Disturbances, Particularly of the Digestive Type, Having a Neurotic Basis.**

It soon becomes apparent to any one engaged in the activities of clinical medicine that abdominal complaints present difficulties far greater than are encountered in any other field to both diagnosis and treatment. In no other region of the body is the evaluation of symptoms so difficult or the solid substance of diagnosis so shifting and elusive as in abdominal disorders. I employ the term "disorders" instead of "diseases" advisedly because of the frequency with which complaints referred to the abdomen prove on investigation to be functional or reflex rather than due to actual pathology within the abdominal organs.

Ill health is usually ushered in by the appearance of symptoms. It is comparatively rare for changes in the size, shape, or consistency of an organ to lead us in the first instance to a diagnosis, and we are accustomed perforce to employ analysis of symptoms to guide us to the detection of organic disease. This may lead us far afield in any case, but nowhere into such blunders as in abdominal diseases. Abdominal symptoms possess little face value and their acceptance at par is most mis-

leading. The reason for this is their singular tendency to overlap, so that they may be referred to other organs or tissues than those pathologically affected. Hardly less confusing is the frequency of functional disorders of the digestive tract arising independently of gross lesions in the organs apparently involved.

In order to better comprehend these vagaries of symptoms arising in abdominal disorders and perhaps acquire some key to their interpretation it may be profitable to consider briefly the peculiarities of innervation of the abdominal viscera.

The nerve connections of the abdominal organs are derived from the autonomic nervous system. Without going into details it will suffice for our purpose to indicate that the functions (sensory, motor, and secretory) of the vegetative organs lie within the control of the two great divisions of that system—the sympathetic proper and the parasympathetic or extended vagus. The groups of fibers forming the sympathetic and parasympathetic are derived, the former from the dorsal and two upper lumbar segments of the spinal cord, and the parasympathetic from the third, seventh, ninth, and tenth cranial nerves and the second and third sacral anterior roots. These two divisions of the autonomic system so constituted are separated by the cervical and lumbar enlargements of the cord which are devoted to the innervation of the somatic muscles or sensorimotor apparatus of the arms and legs. The arrangement of the somatic nervous system is especially adapted for localized accurate reflexes, whereas the visceral system is for more widespread diffuse effects. In the arrangement of the sympathetic we see adaptation to produce as easily and speedily as possible generalized effects. The parasympathetic is less widely spread in its arrangement, resulting in more strictly localized control.

The autonomic system, as its name implies, is forever beyond the control of the conscious will. Its reaction to stimuli consequently cannot be voluntarily controlled. Though we may deaden the stimulus by conscious control, we cannot prevent or inhibit the response once it is evoked. Under normal conditions we are not clearly conscious of our internal organs, whereas the specific sensations aroused by stimulation of the



sensorimotor apparatus and special sense organs are the source from which our consciousness is derived. Although secretory processes and movements of the alimentary tract and its appendages do not usually pass the threshold of consciousness, our internal sensations nevertheless send impressions to the brain which undoubtedly color our individuality, and we may become aware of any unusual change in them. Hence they may play a part in producing discomfort and depression on the negative side or a sense of well-being on the positive side (Brown). The threshold of conscious perception is, however, not fixed or invariable. It may become altered considerably in individuals of certain predisposition or training. Thus, the so-called "neurotic" learns to speak of his internal sensations with a familiarity and intimate knowledge to which the normal healthy man is a stranger. In the normal state, if internal sensations become intense enough to force consciousness, they excite pain. Neurotic individuals, however, may experience or claim to experience a great variety of sensory phenomena falling short of actual pain.

It would take too long to consider even in broad outline the physiologic activities of the autonomic system. A fleeting glimpse of their influence on body function may be gained by observing the effect of sympathetic and vagus stimulation. Stimulation of the sympathetic results in very wide-spread effects which are strongly co-ordinated. The pupils dilate to increase perception to light, the heart-beat is accelerated and augmented in force to supply somatic muscles with blood, blood-pressure is raised, sweating cools the body, secretory and motor functions of digestion are inhibited, sugar is mobilized in the blood, blood coagulability is increased, the thyroid and adrenals are stimulated to increased function. The combined result of these wide-spread effects has been interpreted as the activation of the body for a struggle and to increase the power of defence.

When we turn to the parasympathetic or extended vagus system we find that stimulation produces contraction of the pupils diminishing perception to light, slowing of the heart-beat

and prolongation of diastole allowing the heart to rest, the production of hunger with secretory outflow of salivary and gastro-intestinal digestive fluids, increased motor tonus in the stomach and bowels. By contrast we see that the reactions of the sympathetic and the parasympathetic systems are strongly opposed. The sympathetic mobilizes the activities of the body, converts potential into kinetic energy, and temporarily inhibits the processes of nutrition. Its functions are, consequently, katabolic. The parasympathetic, on the other hand, performs the service of building up reserve by presiding over the functions of digestion and excretion and resting the heart. Through its agency the body is maintained and fortified against times of need and stress. Its functions are anabolic or constructive. Many of the viscera are innervated by both systems, and where this occurs, their actions are antagonistic. Thus, the sympathetic dilates the pupil, the vagus contracts it, the vagus slows the heart, the sympathetic quickens its beat, etc. So we see illustrated what every biologist and physiologist knows—that balance between opposing forces in the organism is an essential of its existence. This is known to be true of the endocrine glands, and there is good reason to suspect that every portion of the whole body influences every other portion either for good or ill. Stimulation and regulation are apparently in some degree a function of all tissue. In no mechanism is this so plainly evident as in the autonomic functions, and they exert a profound influence on the welfare and functional good health of the individual.

Developing this point further in the interests of the subject under discussion, we find at hand many interesting interpretive observations from the physiologic laboratories. More and more it is appearing that in man and in most of the higher animals the springs of action are to be found in the influence of the stronger emotions which express themselves in characteristic acts and profound alterations of the body mechanism. Many surface manifestations resulting from the emotions are familiar to all (pallor, sweating, tremor, dry mouth), but there are other organs and tissues hid out of sight in the interior of the

body that do not reveal so obviously disturbances of their function attending upon states of intense feeling. Among the organs most sensitive to disturbance originating in this manner are those concerned with digestion.

Emotions attain expression through discharges along the neurons of the sympathetic. All the bodily changes that occur in the intense emotional states, such as fear, anger, and pain, develop as the result of activity in that division, and are in the highest degree useful in the struggle for existence. These perturbations seize and dominate the organs that in quiet times are controlled by the vagus. Thus are reserves mobilized and energy discharged through sympathetic activation, vagus anabolism being meanwhile submerged. This explains the evident antipathy between the emotional states that normally accompany body processes. It becomes plain why hunger is banished and digestion inhibited by fear and grief, why sexual passion is suspended by anxiety.

Emotional stimulation of the sympathetic and its coadjutors, the adrenals and thyroid, means the spending of reserves in the struggle for survival. If unduly prolonged exhaustion may result. As Brown remarks, "A vicious circle—sympathetic dissociation—is the pathologic equivalent of a prolonged struggle."

In the huge laboratory of civilization we see these biologic principles strikingly illustrated in the condition of society today. During the years just prior to 1914 there occurred what appears in retrospect and by contrast to have been the high tide of anabolism. Such sympathetic activation as arose from sporadic economic or social rebellion was submerged by predominant vagus inhibitory influences and prosperity became a surfeit. The advent of war on so huge a scale in 1914 rallied the world's forces of defense. Such stimulation of the sympathetic defense apparatus completely submerged vagus anabolism—spending with no thought of reserve. Undue prolongation of the struggle and exhaustion of reserve without replenishment has brought about paralysis of regulation—sympathetic dissociation in the body of human society. Extending the analogy, we may

look upon the social and labor unrest, low moral tone, greed, abandonment of valued traditions, absence of thrift, and other postwar phenomena as constituting a sort of social neurosis the pathologic equivalent of a struggle too prolonged.

A profound or major emotion marshaling all the defensive mechanism is not the only one which is capable of disturbing bodily equilibrium. Milder affective states, such as worry and anxiety, may check or modify vagus functions. Such emotions if unduly prolonged are capable of beating down a broad pathway through the nervous network; a path of least resistance. We catch a glimpse of this mechanism in the influence of depressing and exhausting nervous strain in producing such functional disorders as high blood-pressure, diabetes insipidus, certain forms of thyroidism, and particularly digestive disorders.

The most striking feature of the evolution of man is undoubtedly the centralization of control in the brain. The higher nervous centers so-called are, however, embryologically much more recent in development than the autonomic nervous network which we possess in common with other vertebrates. Evolved in the subconscious sphere this system remains beyond the control of consciousness, but nevertheless is ever on the alert to protect the individual, and through him the race by instinctive reactions which enable him to successfully evade the dangers which beset his path. We may assume that with primitive man these were mainly physical perils. Modern man, through the complexities of life, finds himself physically secure, but beset by a multitude of unsubstantial threats and troubles that appear to threaten his security. The restrictions of custom and environment convert the once instinctive savage reaction to danger and opposition into worry, perplexity, and distress of mind. The autonomic reactions to these abundant emotive stimuli, while they may appear controlled, are never suppressed. Crile has likened the worried and harassed neurotic to an engine running full speed with the clutch out. Motion and speed there is, but it is internal and not converted into its equivalent of physical action. The wear on the machinery is great and breakdown is inevitable if it be too long continued.

Before we shall be in a position to understand the way reflex abdominal symptoms come about we must take into account certain important peculiarities of the reflex arc in the abdomen. The general sensorimotor nervous system consists of parts of neurons the cells of which lie within the brain or spinal cord and they respond to stimuli at once, the delay being measurable in small fractions of a second. An important difference exists in the constitution of the autonomic apparatus. This system is made up of neurons reaching out from the brain or cord and they are not distributed directly to the viscera, but are gathered into ganglia or "transformers" which are believed to modify the impulses received from the central nervous system and adapt them to the peculiar more slowly acting tissues secreting cells and visceral muscles to which the postganglionic fibers are finally distributed. By reason of this arrangement the response to stimuli is in the autonomic system slow and diffuse rather than rapid and local, the delay being measurable in seconds. This explains what Head pointed out, that when a painful stimulus is applied to a part of low sensibility (slow visceral reaction), which is in close central contact with a part of much greater sensibility (cerebrospinal), the resulting pain is referred to the latter rather than to the former, where the stimulus arose. In consequence of this the pain of visceral disease is often felt at a point distant from its place of origin. The internal organs are not themselves sensitive to painful stimuli, but they can cause pain by reflex stimulation in the central nervous system of nerves supplying other tissues to which parts the pain is referred. Another important consideration is that the ramifications of the abdominal network result in each organ influencing reflexly other organs, and if the reflex action be sufficiently strong function may become perverted in the organs so affected. As a result of these nervous reactions and interactions it often comes about that long before any change can be detected in the structure of an organ, reflex sensory and functional symptoms have been in evidence.

Passing now to the practical bearing of what we have been considering, I think you will agree with me that the most fre-

quent complaints we are called upon to prescribe for are abdominal and particularly digestive. So great, indeed, is the number of dyspeptics and so persistent and disappointing their efforts to secure relief that the law of supply and demand has operated to call into being a subspecialty in medicine known as "gastro-enterology." Whether this is working out for the benefit of the public or the good of our profession must remain a matter of personal opinion. All evolutionary analogy in the physical world appears to prove that beyond a certain point specialization is gained at the expense of adaptation, which is a much higher essential. Adaptation in its medical bearings implies a continuous and close touch with fundamental principles combined with capacity for sympathetic understanding in the interpretation of all aspects of disease. Overspecialization unquestionably narrows the perspective and restricts the outlook to a smaller schedule of interests. The gastro-intestinal invalid may comprise within his disordered mechanism many considerations that cannot be gaged by test-meal, tubes, or x-ray. Nor can he always be relieved of his discomforts or solaced of his fears and apprehensions by special dietaries, so-called "cures," or operations. The very name "gastro-enterology" implies to the patient a diagnosis already made and confirmed, and when we see how far afield this may lead the functional invalid and what therapeutic vagaries the stressful ingenuity of this new specialty creates a doubt may well intrude regarding this departure in specialism.

Let us consider for a moment the most common systematized digestive complex that is brought to our attention—the so-called "ulcer syndrome." Ordinarily the complaint is of pain or painful discomfort referred to the abdomen. This may or may not possess a certain time relationship to the taking of food. There is usually more or less evidence of indigestion, heartburn, eructations, belching, and commonly constipation, with more or less addiction to laxatives. Many of these patients display abdominal operation wounds. When examined physically a more or less definite tender point is elicited or the belly may be diffusely tender. The tender point when present

may be in the epigastrium or right hypochondriac or iliac region, or over the operation scar if one exists. After the employment of examination methods—clinical, laboratory, and x-ray—to determine whether an ulcer actually exists we have found in our hospital service that from 10 to 15 per cent. have undoubted ulcer, the remainder being grouped as colitis, reflex dyspepsia, spastic constipation, etc. Undoubtedly we often fail to properly classify these patients, but few ulcers escape detection. The complete gastro-intestinal x-ray examination determines these cases accurately and with a very small percentage of error.

Fortunately, for my purpose, we have available at this point a most interesting and instructive statistical analysis of a large group of patients of this type. In the Journal of the American Medical Association for October 29, 1921 Blackford publishes an analysis of 1000 cases. Extracting from his summary of conclusions such data as are applicable to our discussion we find that 14 per cent. of the 1000 patients actually had organic gastric disease, and of these, 9.9 per cent. had peptic ulcer and 3.8 per cent. carcinoma. In strong contrast to this he found 34 per cent. showing extragastric abdominal disease giving reflex dyspepsia. Of this 34 per cent. reflex digestive disorders inflammations of the gall-bladder accounted for  $15\frac{1}{2}$  per cent. and the appendix for 7.8 per cent.; constipation for 7 per cent.; 18 per cent. was answerable to systemic disease, such as syphilis, tuberculosis, nephritis, primary anemia, etc.; 25 per cent. of the total 1000 cases presented no organic pathology and were classified as functional.

While perhaps it may be unfair to be too arbitrary in drawing conclusions from a single series such as Blackford presents, the testimony it affords is apt to give one a guilty feeling when his own experience is passed in review. The stomach has been called "the greatest liar in the body." It has also been named "the spokesman of the abdomen." Which of the two implications we accept will be determined by our understanding of the reflex mechanism which operates in the abdomen. Rightly understood gastric symptoms may as often as other manifestations help us to make a proper diagnosis. Abdominal diag-



nosis is a great leveler. Where we all err so frequently no one is in a very good position to criticize. The reason for our failures perhaps more frequently than for other reason lies in our omission to secure a full and exhaustive clinical history, and to make a proper physical examination with our attention alive to the peculiarities of abdominal sensory vagaries. We should at least thereby be able to guard against the mistake of treating a patient for ulcer who has an incarcerated inguinal or umbilical hernia, of operating upon the abdominal neurotic with dropped organs and irritable colon, or the tragic error of opening the belly of a patient whose pain represents the gastric sensory crisis of tabes. An unprejudiced lay observer might conclude that as a profession we entertain a very poor opinion of the human stomach. He would be led quite legitimately to this conclusion by the manner in which drugs, tubes, and special dietaries are employed when the dyspeptic takes his complaints to the doctor. As a matter of fact, the stomach is singularly resistant to disease. It is more sinned against than sinning, and unless it develops a new growth or is injured by swallowing noxious material it proves man's one faithful organic standby, enduring all kinds of abuse without rebellion until life is forfeited through the failure of another less potential organ. When dyspepsia appears the stomach is not usually at fault at all, but is worried by an irritable focus elsewhere.

An amazing fallacy that in recent years appears to have taken hold of internists and surgeons as if it were dictated by fashion is that every patient with pain in his abdomen, hyperacidity, and a tender point is guilty of having peptic ulcer unless he can be proved innocent. This quaint idea appears to have entered the medical mind with the epigram of a brilliant surgeon that "hyperchlorhydria is ulcer masquerading in the medical wards." Without regard for the verities someone piled error on error by stating that pain relieved by eating or by soda meant ulcer. Ulcer has certainly experienced a boom, for now we have ulcer clinics where are gathered hitherto unheard of numbers of patients being treated for that disease. Any dyspeptic seeking distinction in his world must nowadays be able to boast of an

"ulcer cure." It bids fair to displace the vogue of chronic appendicitis. And the wonder is that this has taken place co-ordinately with the development of x-ray technic that has immensely clarified the obscure problem of classifying abdominal disturbances. Although perhaps in other times we might look with tolerance on a certain liberality in symptom interpretation, we are today in a position to insist on certain criteria (x-ray and other) before diagnosing the case as ulcer. If this were always done the number of patients subjected to ulcer treatment would, I am sure, be less than it is. At this point a brief discussion of so-called "chronic appendicitis" as a factor in chronic abdominal disturbance may not be out of place. This is a condition much more familiar to the surgeon than to the medical man. I have heard it told of an internist of national fame that when invited to open a discussion on chronic appendicitis, he replied, expressing his regret at not being qualified to do so, as he had never seen a case. I have never yet heard a satisfactory definition of what chronic appendicitis really is. Undoubtedly, it does not require much pathology in the appendix to give rise to reflex disturbance of digestion, yet I have seen many grossly normal appendixes removed at operations for "chronic appendicitis." In Blackford's series 13 per cent. of all patients complaining of chronic digestive disturbance had lost the appendix before coming to the clinic. The failure in diagnosis in this class is evident, since the patients were still complaining of digestive symptoms. There is one type of patient in particular where we almost expect to find one or more operation scars in the lower abdomen. This is the constitutionally inferior thin-bellied woman with dropped organs and spastic colon. Ricord once defined woman as "a creature who is constipated and has a pain in her side." She of the type is tender over the cecum, where a burning pain may be located, and she is operated to remove the pain supposed to be due to the appendix. The appendix is found long, kinked, constricted or clubbed, and the diagnosis and operation appear vindicated, yet the pain persists. Subsequently further surgical effort is often forthcoming to relieve "adhesions," with similar disappointing results,

and even this may not end the unfortunate woman's surgical experiences. I have seen as many as four abdominal operation wounds on such a patient. A similar tale might be told regarding suspension operations now fortunately losing their vogue. It seems to me that a diagnosis of chronic appendicitis should not be entertained unless there is a definite history of a previous acute inflammatory attack. If we kept always in mind what we know or should know regarding digestive reactions in general and reflex abdominal symptoms in particular we would make fewer mistakes in diagnosis. Modern x-ray technic is of the greatest value in identifying the cause of reflex abdominal disturbance. Not only may the presence of ulcer be determined in a high percentage of cases where it exists, but gall-bladder disease, malignancy, static intestinal conditions, diverticuli, and other important factors often stand revealed. No less essential is the general physical examination. Chronic heart and kidney lesions, central nervous disease, tuberculosis, chronic oral and nasopharyngeal infections, anemias, intestinal parasites, hemorrhoids and anal fissure, kidney and ureteral stone, pelvic infections and neoplasms, and many other discoverable conditions, to say nothing of the habitus of the patient, enter into the proper evaluation of abdominal symptoms and may furnish an adequate explanation therefor.

To close this decidedly random discussion of a subject having many important ramifications, let us revert to the practical bearing of what was said early in my remarks about the influence exerted by disturbing emotional states on the autonomic nervous system and the secondary effects thereof. You will recall that reference was made to the profound effects observed experimentally upon the motor and secretory functions of the stomach and bowels as a result of major emotions such as fear, rage, and pain operating through the autonomic mechanism. These wide-spread and highly co-ordinated reactions are regarded by physiologists as phylogenetic in significance, as a response by the organism to that most primitive and most important instinct of self-preservation. The preservation of the individual obviously must precede the preservation and perpetuation of

the race, and it appears, therefore, to be the basic instinctive impulse of mankind as it is of creatures lower in the scale.

Man is no longer beset by physical peril as in his primitive state, and except in time of war and the rare and usually accidental occasions of great danger his life-saving autonomic mechanism is rarely called into full play. There is little doubt, however, that the anxieties, worries, griefs, and disappointments of complicated strenuous present-day existence are in some measure activating and setting in motion his autonomic safeguards. There are certain individuals whom we loosely class as neurotics who appear to be keenly sensitive to influences operating in this manner. They do not conform to any physical type that lends itself to description, but they possess in common certain temperamental peculiarities which serve to identify them. Chief among these is their liability to develop what we may call "fatigue reactions" under strain, whether it be emotional or physical strain, but especially the former. The threshold of consciousness becomes lowered when their reserve is depleted by unusual demands, and they react to sensory stimuli ordinarily submerged. You are all familiar with such manifestations in these individuals as fatigue backache, and headache, with the palpitation, cold hands, and rapid pulse, which of late has become known as "neurocirculatory asthenia," and especially common is nervous dyspepsia with spastic constipation. Patients belonging to this group usually have lost more or less of their physical reserve power and sense of well-being and complain of malaise, lassitude, and other lack of fitness as well as of their abdominal sensory distress. Long-continued causes of fatigue may give rise to persistent manifestations of digestive disorder for which diet control and drug treatment appear ineffectual. As a rule, these patients are persuaded by the chronicity of their symptoms that there is some organic disease present. They very often are highly suggestible and apprehensive, and take alarm when an erroneous diagnosis or the prescribing of drugs appears to confirm their suspicions. The reading of magazine or newspaper dissertations on disease may lead them to fit their symptoms into a description of some serious organic

malady. They are not in a position to know unless it is explained to them that their sensations are such as occur normally, but are not normally cognated or are ignored by the normal mind. In certain of these individuals the abdominal history begins with some transient visceral inflammatory attack or some functional upset resulting from ordinary causes, or perhaps with some abdominal operation necessitated by an acute infective process. In either event the presence of symptoms trains the attention of the patient to observe physiologic processes, in the course of time developing an introspection that makes him increasingly sensitive to sensory stimuli. Presently this becomes a **fixed habit** leading to **unconscious reproduction** of symptoms long after the initial cause has disappeared.

We may see in developments of this character that the more excitable the nervous system grows, the lower becomes the threshold stimulus required to cause symptoms, and, on the other hand, in individuals of a certain predisposing temperament symptoms at first purely legitimate may, by disturbing perception centers, perpetuate themselves so as to constitute a neurosis. Bearing the foregoing in mind, we should, while carefully avoiding any attitude that might confirm his fears, endeavor to put the patient's mind at rest by tactfully explaining to him the true nature of his complaints. It will not suffice to tell him brusquely that there is nothing really the matter with him. It is a problem calling for instruction and persuasion. Above all, surgical operations on neurotics, and particularly on those displaying the asthenic habitus, should be avoided unless absolutely necessary. Fortunately, thoughtful surgeons realizing this are more and more restricting their operations to definite organic lesions.

## CLINIC OF DR. CHARLES SPENCER WILLIAMSON

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### PERNICIOUS ANEMIA

#### **Presentation of 5 Cases of Pernicious Anemia Representing Various Types and Stages of the Disease. Diagnostic Hints.**

THE cases which I wish to present to you today are all examples of one and the same disease, about which a good deal has been written lately. They are cases of pernicious anemia, and you are doubtless aware there have been in the last year or two many publications dealing more especially with the subject of treatment. I regret to say that while our treatment has improved to such an extent that we may perhaps offer a patient suffering with this disease some amelioration, yet the outlook for the patient who really has this disease is now just what it was years ago. I want to approach the subject with you from a purely clinical standpoint and show you under how many different guises the disease presents itself. It just so happens that we recently have had an unusually large number of patients, and I have brought from private practice 2 or 3 patients who are here today. Perhaps the simplest way will be to show you these patients and then later on to discuss them in a little greater detail.

#### **CASE I**

The first patient is a man sixty-nine years of age, who is, as you see, in very fair nourishment and looks rather young for his years. He is an officer in a manufacturing establishment which necessitates his traveling more or less, and when he is at home, which is in an adjoining state, he carries on rather onerous business duties. I was first called to see him twelve years ago,

at which time he was desperately ill and it was hardly thought that he would live more than a few days. In consultation with his local physician I spent the entire day making as elaborate an examination as was possible in that time. The notes which I have of his case as they were given me by his family physician at that time are, briefly, as follows:

Six or eight months previously the patient, who had been in excellent health up to that time, commenced to develop trouble with his stomach in that he had dyspeptic symptoms of a rather indefinite type, but quite severe. He would have pain at varying times after eating, and little by little nausea and vomiting developed, which were sometimes independent of food taking and sometimes followed it—an hour or an hour and a half afterward. There had never been any blood in the vomitus, but he had had occult blood on several occasions in the stool, being, of course, on a blood-free diet when this examination was made. A number of test-meals had been given him, and they gave uniformly the same result, namely, that he had a total achylia, both the hydrochloric acid and ferments being absent. An x-ray examination had not been made, and the vomiting was so pronounced when I saw him that it was inadvisable to make such an examination. His red cells were down to about a little over 2,000,000, but showed nothing especially significant. The hemoglobin was diminished in about the same proportion. The whites were approximately normal.

Physical examination by his two local physicians and myself failed to reveal anything abnormal except that he had a definitely palpable spleen. We went into the pathologic history in great detail, and could find absolutely no reason for this enlarged spleen along the line of any previous disease; that is, there was no antecedent malaria or typhoid or any other infection which could legitimately account for the enlargement of this organ. I call your very especial attention to this point, because it was the one thing which prevented me from accepting the diagnosis of carcinoma, which had already been made. As it was, we considered pernicious anemia, but when I left our diagnosis was still in doubt.



To make a long story short, under careful feeding of small quantities of liquids at short intervals and gastric lavage the patient shortly after began to improve, and in the course of three or four months was up and about and apparently in as good condition as before he had been taken sick. He went away on a trip to the Bermudas, and called to see me in Chicago on his return. I should not have known him. He wished an exhaustive examination made, and this I did for him. I could find absolutely nothing abnormal except that he still had his achylia. The spleen could no longer be palpated.

Since this time I have seen him two or three times every year at the very least, sometimes for minor troubles and sometimes because he wished me to examine him, for he has been quite apprehensive about his condition during all this time. I have made, I suppose, a dozen gastric analyses in the last ten years, with always the same result. One symptom, however, has been worthy of note. On small provocation he would develop a diarrhea and under circumstances when it could not be intelligently ascribed either to indiscretions in diet—and parenthetically I may say that he is a man who takes most excellent care of himself—or to sudden changes in weather, exposure, or causes of that kind. These attacks of diarrhea would last from three or four days to a week. Rest in bed with a diet consisting of boiled milk and cereals checks it promptly. During all this time he has been taking fairly large doses of dilute hydrochloric acid, and failure to take this almost invariably results in the development of a diarrhea, so much so that he looks upon this hydrochloric acid as a necessary part of his daily menu.

Our patient continued in good condition until about a year ago, when he began to develop a marked weakness with moderate loss of weight, and this weakness progressed to such an extent as to more or less interfere with his business duties. Consulting me at this time, I found that his red cells, which had averaged well over 5,000,000 during all these years, had suddenly gone down to a little over 2,000,000, the hemoglobin being reduced in somewhat less degree so that the color index was

1.2+. The spleen was again palpable. Histologic examination of the blood showed a considerable macrocytosis, especially of the large oval forms, which are, and I think correctly, given a place of considerable importance in the diagnosis. The white count showed a definite leukopenia with a predominance of the lymphocytes.

During all these years I was in doubt as to the original diagnosis. A fairly large experience with pernicious anemia made me conversant with the fact that achylia or, at least achlorhydria, is a sign which develops very early. I have come to look upon it as almost one of the cardinal signs of the disease. A sudden drop in the blood with absolutely no infection of any kind to account for it, no focal infection discernible anywhere, led me to conclude even then that the diagnosis of pernicious anemia, which we had only tentatively made years before, was correct. The patient was put to bed and it was only with considerable difficulty that a remission was brought about. The blood stayed around 2,000,000 for some time, but gradually began to improve, and in the course of three or four months, much of which time was spent in bed under active treatment, it reached 4,500,000 again.

Then for about a year, that is, up to quite recently, he remained in fair condition, always, however, having a palpable spleen and always feeling, as he expressed it, "rather washed out." A few weeks ago he consulted me again, and I found that his blood had taken another turn for the worse, so that he now has a trifle over 2,500,000 red cells, the hemoglobin being 10 grams per 100 c.c., and again a leukopenia with a predominance of the lymphocytes. There can be now not the slightest question as to the diagnosis. When we sum up the case we shall see one striking thing, namely, a duration of over ten years between the first relapse and the second. During all of this time the stomach findings were present, but no other symptoms which could be in any way ascribed to the disease. I neglected to say that at the present moment he is complaining rather bitterly of pains in the arms and legs, with numbness and tingling in the fingers and toes. While the objective changes

are slight, the knee reflexes show a definite diminution, and there can be little question that he is developing beginning cord symptoms, although these were never present previously.

Let me emphasize again the fact that the patient had an achylia and that occult blood was present for some time in the stools, leading, not to our surprise, to a diagnosis of carcinoma of the stomach. Note especially the extremely long remission with almost perfect health, and then finally the relapses, in one of which you now see him. Remember that the achylia was present during all of this time.

#### CASE II

The next patient is a woman forty-two years of age, also a private patient, who consulted me in March, 1919. She was a maid of one of my patients. Her employer asked me to look her over because she looked so white and pasty and seemed so apathetic and mentally dull. The notes which I made on the patient at that time are as follows:

The patient had been complaining of stomach trouble for four or five years, manifesting this in a dislike for food, eructations of gas, moderate discomfort after eating, which, however, never amounted to actual pain. Vomiting occurred only occasionally and particularly, she thought, after taking fatty articles of food.

Physical examination showed a striking lemon-yellow tint. She was distinctly emaciated and stated that she was 35 pounds below her normal average weight. She had a beginning edema of the ankles and very marked shortness of breath, and a little tenderness over the liver. This may have been an extremely low degree of venous congestion of that organ, although neither physical examination nor the x-ray revealed any cardiac dilatation. The spleen was definitely palpable about an inch below the edge of the ribs and was quite firm.

The patient is a Swede and has spent some time in Minnesota, and for these reasons an especially careful search was made for the eggs of the *Diphyllobothrium latum*, but none were found. When she first consulted me her red cells numbered

500,000 and the leukocytes 2300, with a predominance of the small lymphocytes. Macrocytes were abundant and the blood-picture was as typical as one could possibly ask, so much so that the diagnosis could have been made with a high degree of certainty from it alone. She had had no infections which might cause an acute anemia.

For various reasons the patient was unable to enter the hospital at this time, and it was nearly six weeks before she actually was in the hospital under my care. During all this time she had been growing definitely worse. On admission she had lost 5 or 6 pounds more, the edema was quite marked in the lower extremities, and a careful blood count showed the reds reduced to 450,000, one of the lowest counts which I have personally ever seen. It was checked up quite carefully. She had 2.3 grams of hemoglobin per cubic centimeter, and according to my tables the average for that age is 15.64 grams per 100 c.c., making a color-index of about 1.5. The test-meals showed a very great reduction in hydrochloric acid, amounting almost to an achlorhydria, the ferments, however, being present.

The patient was treated subsequently by rest in bed with forced feeding to the limit of her capacity to digest food. She was given iron and arsenic subcutaneously. I mention this, although personally I am exceedingly skeptical as to the value of iron therapy in this disease, especially in cases where the color-index is 1+, showing that the individual corpuscles have already a greater load of iron than normal. She spent about three months strictly in bed and showed a striking improvement almost from the first day. In approximately four months she had gained over 30 pounds, and the red corpuscles shot up with great rapidity until they reached a point just a little under 5,500,000, many times what they had been in the beginning. It is interesting to note in this connection that the color-index, which had been 1.5 in the beginning, now became markedly reduced, it being only 0.7. This is easily accounted for when one remembers how frequently in other conditions the red corpuscles outstrip the hemoglobin in rapidity of regeneration. Although improvement in hemoglobin was striking, it lagged

behind the red cells, so that, as already stated, the color-index became minus. In all other respects the blood seemed perfectly normal. I think that had anybody seen her now for the first time the diagnosis of pernicious anemia would have been absolutely impossible.

I am showing you this patient because in my experience she is almost unique. To begin with, the blood-count originally had reached an extraordinarily low figure; second, in my experience—and I believe I am correct when I say that this is the experience of most authors—it is very rare for a blood-count which goes down even as low as 1,000,000, to say nothing of below 500,000, to ever again reach such a mark as 5,000,000, even though the remission is most marked in other respects. It is not very unusual to have a count of 2,000,000 go up as high as 3,500,000 or 4,000,000 during remissions, but in a fairly large number of cases which have come under my observation I have never seen or known of a remission of such an extent as this. I doubt if the small amount of arsenic the patient received had anything to do with it. We gave, as we always do, considerable quantities of food rich in iron, such as beef-juice and egg-yolk, but these could do little more than furnish the iron, and by no stretch of the imagination could they be held responsible for the clinical improvement. (Sends the patient from room.) I have sent the patient out because the after-history is the usual one. The remission lasted only a few months, and from then on she has had several sharp relapses with moderate remissions. During this time she has been partly under my care and partly under the care of others, but I have kept in touch with her constantly. At the present moment, as you see, she is in a most distressed condition. A blood examination would not be of great value just now, since she has recently had a number of massive transfusions, but in spite of these she is steadily going down hill. Cord symptoms have developed and the case is now an outspoken one of pernicious anemia in the last stages, for I do not think it possible that the exitus can be much longer delayed.

Let us emphasize the salient point of this case, which is

the remarkable degree of the remissions, the blood going from below 500,000 to a point nearly 5,500,000, which is well above the average for the normal woman.

### CASE III

The third patient I want to show you represents again a different type of the disease, in that this is one of the examples of pernicious anemia, or at least that is our diagnosis, in a boy of barely eighteen. He came into the hospital on January 9th with the following history:

He has been exceedingly weak and has been pale for the past eighteen months. He was working on a farm in Ohio and became gradually quite constipated, for which trouble he took Epsom salts regularly, beginning about June, 1919. He thought very little of this constipation and kept on working until August. In October of that year he was in the hospital for two weeks, and his physicians at that time told him he had pernicious anemia. During these two weeks he was too weak to be up and around. He came to Chicago in February, 1921 and got a light job, which he kept until March, 1921, at which time he had another very severe attack of weakness and was in the hospital for four weeks. Since that time he has had no definite attack, but has noticed that he has been getting weaker, especially during the last few weeks.

We see, then, that he gives a very clear-cut history of a relapse which lasted two weeks in October, 1919, with a second attack in March, 1921. Oddly enough, and contrary to the usual state of affairs, he has noticed that his bowels do not move during the times when he is the worst. In March, 1921 there was a period when he was so weak that he could not turn over in bed. The pallor was noticed in 1919. It then cleared up with the improvement in his condition and returned again during the second relapse. Later it improved again somewhat, but not as much as it did at first. During the last month he has been getting exceedingly pale. He is not quite sure about his weight. He thinks he has lost some weight, but not a great deal, and judging from his appearance I should say that it is

certainly not very much, if any. He now complains of marked shortness of breath, especially on exertion, and this he had during both previous attacks. For the last month he has complained bitterly of dizzy spells which are very marked, and which he attributes to his weakness, especially when he tries to get up and go around. He has never had a sore mouth or tongue and has never had any diarrhea, but, on the contrary, he has been constipated at times when he has been worse.

His pathologic history shows nothing of consequence except that he had pneumonia at eighteen months and mumps and measles at fourteen years.

His family history shows nothing having a bearing upon his present condition.

*Physical Examination.*—This reveals a young adult male, about eighteen, having a distinctly lemon-yellow tint, and who does not seem acutely ill, but exceedingly languid. This note was made on his admission to the hospital about six weeks ago.

The eyes, ears, nose, and throat were normal. Lungs and heart showed nothing except a slight systolic murmur at the apex. The abdomen was negative, save that the spleen was enlarged to a point about three fingers below the edge of the ribs. The liver was not palpable when he came, but I may say that now it is definitely enlarged and palpable, about one to two fingerbreadths lower than normal.

Examination of the extremities and especially the bones, reveals no tenderness or abnormalities, with the exception of several scars which followed abscesses where he was given iron injections last spring. The reflexes were normal and his temperature on admission about 100.4° F., around which point it has stayed ever since he has been in the hospital.

On examination the urine showed a trace of albumin. Test-meals showed the absence of hydrochloric acid, the absence of organic acids, and almost a negative total acidity. The Wassermann was negative.

The blood examination, which was made repeatedly and with great care, showed 1,400,000 erythrocytes, 33 per cent.



hemoglobin, and 1900 leukocytes, with a color-index of 1.18. A differential count of the whites shows small mononuclears, 69; large mononuclears, none; polymorphonuclear neutrophils, 25; eosinophils, 6, with macrocytes and microcytes present in large number. The poikilocytosis was quite marked and 1 megaloblast was noted.

His career since he entered the hospital has been steadily downward. He has been transfused with fairly large quantities of blood five times between January 16th and March 1st, in spite of which the blood-count has never even attained the 1,400,000 which it showed on entry. It has been around 1,100,000 all the time. At the present time he is running a higher temperature, it averaging approximately 102° to 102.5° F. On February 1st we considered the question of the entire condition being an acute infectious hemolytic anemia, and blood-cultures were made, with, however, absolutely negative results. Pernicious anemia is such a rare disease at this time of life that it was with great difficulty that we have been able to make up our minds to make this diagnosis, but I think you will all agree with me that at the present time it is scarcely possible to make any other diagnosis. He is developing a loud, harsh murmur over the pulmonary area which is readily explainable on the basis of his anemia. Cord symptoms in the nature of foot-drop and some inco-ordination have developed and he is complaining of cramps in the lower extremities. The edema of the legs, as you see, is now quite marked, and there is a bed-sore which has developed over his sacral region, for in the last few days he has developed incontinence of urine and feces.

Reverting for a few minutes to the diagnosis, I want to call your attention especially to the condition I referred to a moment ago as that of acute infectious hemolytic anemia. This is a condition which in my judgment is not sufficiently appreciated, although it does not constitute a definite clinical entity. It is not even very widely known that a number of acute infections of septic nature, particularly streptococcus infections, can produce an anemia which will depress the number of red cells from 5,000,000 to as low as 2,500,000 or even 1,000,000

in a comparatively few weeks. I have seen this in puerperal sepsis on two occasions and several times in acute streptococcus septicemia from other causes. I do not now refer to those cases of subacute septicemia such as we so frequently see in endocarditis, for example, due to the *Streptococcus viridans*, but to a much more acute process. We must think of this very strongly in the patient before us. The mere fact that he had a diagnosis of pernicious anemia made a year or two ago goes to show that his condition at that time was very bad, and presumably a blood-count was made which seemed to justify such a diagnosis. On the other hand, he must have improved materially or he would not have been able to come to Chicago and go to work again, even though he had a light job. Then, the second relapse and the second remission are, to my mind, most important reasons for considering this a case of pernicious anemia rather than considering these previous attacks some other condition and the present one an acute hemolytic anemia. The blood examination is not typical of those obtained in the acute infectious anemias. The enlargement of the spleen I want to lay great stress upon because of its great diagnostic value. I do not think sufficient attention is paid to this feature of the clinical picture of pernicious anemia. It is commonly stated that about a third or a half of the cases have enlarged spleens. My own personal experience has been that a somewhat higher proportion, fully half, will disclose a palpable spleen at some time in the course of the disease if it be carefully looked for, and, further, when found, it is of the very greatest importance. Unfortunately, in making a differential diagnosis between an acute septic anemia and pernicious anemia with fever this sign is of no value, since the former shows a large spleen as well as the latter.

Our patient is, as you see, seriously sick. I shall show him to you at a later clinic and we shall see what progress he is making.

**Note.**—Clinic a week later. I want to report to you the subsequent history of the young lad in whom I made a diagnosis of pernicious anemia at our last clinic. While we recognized

that he was critically ill, we hardly expected that he would die as soon as he did. He died yesterday. An autopsy was made and the organs I have here are from him. The diagnosis of pernicious anemia which we had made with some hesitation proved to be correct. As you see, the spleen shows a very marked hyperplasia, it being at least three times the normal size and weight. The liver shows a definite cloudy swelling and fatty changes which account for its large size. Beyond this nothing abnormal was found in the various organs. Specimens of the bone-marrow were taken, which will be subjected to further study. You will remember that I discussed at some length the question of an acute infectious origin, and I wish to emphasize this point again, for I am sure that a diagnosis of pernicious anemia is occasionally made when the real underlying condition is an acute septicemia, especially of the streptococcic variety.

#### CASE IV

The next patient represents a still different angle of this disease. He is a mechanic, fifty-two years of age, who came under our observation with three major complaints, namely, diarrhea, pain in the abdomen, and loss of weight and strength. All of these had a duration of from six to seven years.

The onset of the disease began with a diarrhea which came on fairly suddenly and of which he thought very little in the beginning. It was of a fairly severe type and characterized by loose, watery stools, yellowish in color. The diarrhea persisted for three or four months, and he thinks from nine to ten stools a day would be a fair average for that time. Since then he noticed that he has scarcely ever had a day with less than four movements, and they have been averaging, for some time past, six a day. For a considerable period of time they were dark greenish, and five weeks ago he says they became light colored and, he thinks, rather slimy. On being asked specifically as to any periods of freedom from diarrhea he denies any such, stating that he has had it practically continuously since the onset between six and seven years ago.

**Pain.**—This is localized in the lower abdomen, and he de-

scribes it as a feeling of soreness referred largely to the suprapubic region, but more or less over the entire abdomen. He noticed it a couple of days after the beginning of his diarrhea and it has persisted ever since. He thinks it is not especially intensified by bowel movements, although he does experience a sensation which he describes as "rawness" in the abdomen.

**Loss of Weight.**—This was especially marked at the very beginning of his illness. He states he lost 37 pounds in the first two months, and since that time the weight has been substantially stationary, with slight fluctuations up and down. The loss of strength has gone hand in hand with the loss of weight, being most marked in the beginning. While he has continued to work, being forced so to do by his finances, he has had to rest for weeks at a time, and has kept this up until five weeks ago, when he began to feel unusually weak and complain of dizziness.

These are substantially his symptoms now. He has no especial cardiovascular, respiratory, nervous, or genito-urinary symptoms. He has never vomited and has never had any blood in the stools. Jaundice has never been present.

His previous history is unimportant. He has never been a hard drinker and at present drinks none at all.

On admission his temperature was a little subnormal and as an indication of his weakened condition his blood-pressure was 86/42.

The essential features of the examination on admission were that he was badly nourished and cachectic looking, apparently very anemic. The tongue was smooth and glistening, the papillae showing marked atrophy. The teeth were such as are usually found in men in his station of life. The chest showed no abnormalities. The abdomen was distinctly scaphoid and nothing abnormal could be palpated. In particular the spleen was not enlarged. *x*-Rays of the gastro-intestinal tract were all negative. There was edema of moderate degree involving the feet as far up as the ankles and the reflexes were normal. No other abnormalities were noted in any part of the body.

From the laboratory side we note that the urine was normal,

that the feces even after many examinations showed no parasites or ova. The gastric contents showed free acid 0, with a total of 3, there being no blood. Examination of the blood itself showed 2,200,000 red cells, 7400 white cells, and a hemoglobin of 55 per cent. A differential count of the whites showed on the first examination 32 per cent. polymorphonuclear neutrophils, 6 per cent. large mononuclears, 62 per cent. small mononuclears, with macrocytes and microcytes present in considerable number. There was considerable poikilocytosis and much basophilic degeneration.

The progress of the patient while in the hospital has been steadily downward. Succeeding blood examinations showed approximately 1,800,000 reds, and it remained at this figure for many weeks, the leukocytes gradually being reduced to about 3000. It is interesting to note, although I am not clear as to its significance, that the proportion of polymorphonuclear neutrophils increased relatively to the other cells until they averaged over 80 per cent. of the total number of white cells present. The Wassermann reaction was negative.

I will not detail to you the various things that have been done for him except to say that he has had repeated transfusions and the usual hygienic and dietetic treatment. Arsenic has been given, but in spite of all of these things the patient has gone steadily down.

You will notice that we have the patient restrained for the reason that a few days ago he developed a more or less maniacal condition, and his strength is becoming rapidly exhausted from lack of nourishment and from the constant motor activity.

Analyzing the case as it presents itself now, we are face to face with a differential diagnosis involving practically only two things, a secondary anemia due to some gastro-intestinal condition or a primary pernicious anemia in which the long-continued diarrhea has been the main symptom. It must be noted that almost from the time he came into the hospital the diarrhea ceased. This, to my mind, is an important diagnostic point. My experience is that the diarrheas of pernicious anemia

are not at all like the infectious types and are readily controlled so far as the symptom itself is concerned, though the disease progresses just the same, whereas, on the other hand, infectious diarrheas of whatsoever kind, of such severity, are controllable frequently with the very greatest of difficulty. The most careful examination has failed to show anything that could be ascribed as ulcerative colitis or a malignant condition of the bowel or disease of any similar nature, especially parasitic.

A further and, to my mind, quite important sign is the atrophy of the tongue. This is a well-known feature of progressive primary anemia, and I think was one of the things which first convinced me that the case really was one of progressive pernicious anemia.

In view of the steadily progressing anemia and in view particularly of the histologic changes in the blood we are compelled to make a diagnosis of primary pernicious anemia.

My real reason for presenting this patient to you is to emphasize a point which, although very well known to many, is apparently unknown to a large number, namely, that persistent diarrheas without adequate cause call for a careful examination on the basis of the patient having a pernicious anemia. I have seen many cases strikingly similar to this one and one in which many "cures" for various intestinal conditions have been carried out, under the assumption that the diarrhea was parasitic, and yet autopsy showed a primary pernicious anemia. I personally lay considerable stress upon the diagnostic value of an enlarged spleen if present, but we have not been able to demonstrate that in this patient. As he is fairly thin, we should have no difficulty in determining it.

**Note.**—Two weeks later. This patient died rather suddenly at the last, the general course having been steadily downward, and with no associated complications except the increasing degree of hypostasis in the lung. An autopsy was secured which revealed no cause for the anemia, so that the anatomic diagnosis is "primary pernicious anemia." As an unusual finding the spleen was atrophied. The bowel was apparently normal. Our inference, however, that this long-continued and

ultimately fatal diarrhea had as its basis a primary pernicious anemia is substantiated.

You might think that by now you have seen all of the various types of this disease, but such is hardly the case, for our next patient shows a slightly different onset and course.

#### CASE V

He is sixty-five years of age and has been sick eleven months. His symptoms began with a numbness and tingling in the legs, general weakness, and pallor. They came on gradually. He thinks the weakness came first, and this affected particularly the limbs, becoming progressively worse until five months ago, when he showed a considerable degree of improvement, which he attributes to the warm weather and fresh air. He remained in fair condition until six weeks ago, at which time the weakness returned with increased severity, and since then it has steadily become more marked. He thinks his pallor developed at the same time as the loss of strength, increasing in degree until last summer, when it improved very materially at the same time that his weakness improved, and returned when the latter grew worse six weeks ago. Numbness and tingling has been present since the onset, running a course almost exactly parallel to the strength and pallor, improving when they improved and getting worse when they again grew worse. At present the patient is unable to feed himself on account of the lack of feeling in his fingers. The legs show a definite paresis, the patient being able to move them, but the power is minimal. They are described by him as stiff and numb. At the present time he has comparatively little control of the sphincters.

Aside from these symptoms of which he has complained, he states, on further questioning, that he has some dizziness and some shortness of breath. He states further that there was some swelling of the extremities before his entrance into the hospital, but it disappeared after taking some medicine prescribed by a physician. These are the only significant symptoms which we can elicit, except that he has lost 25 pounds in the past eleven months. He thinks he has never had any



fever and, as a matter of fact, he was afebrile on admission and has remained substantially so. He has been at one time a fairly hard drinker, but has not drunk any for the last ten years. Syphilis is denied and the Wassermann is negative on both blood and spinal fluid.

On admission the physical examination, which was substantially the same as at present, now showed that he was a poorly nourished, somewhat emaciated man, and very anemic looking. There was nothing especially abnormal about the head except that he shows, as did our last patient, a typical atrophy of the tongue. The chest is normal, as is also the abdomen, and there is no enlargement of the spleen which can be determined. Sensation in both lower limbs is not objectively impaired. On the contrary, there is a distinct hyperesthesia in certain portions. The motor power is greatly diminished in both legs to such an extent that the patient is just barely able, as you see, to raise the thighs, but he cannot raise the outstretched leg as a whole. There is moderate spasticity on both sides. The muscle sense is definitely impaired. Knee-jerks are exaggerated, as are also the ankle reflexes, there being a definite although slight clonus in the latter. The deep reflexes of the upper extremity are all lively, but scarcely pathologic. The neurologic examination, our intern tells us, was made with a great deal of difficulty because of the hyperesthetic condition of the patient. He was very irritable and did not wish to be disturbed.

From the laboratory side we note that the urine is normal. The stomach contents show free acidity, 0; total, 2, with much mucus present. The stools were negative for blood and parasites or ova. Roentgenologic examination was entirely negative. The blood shows a red count of 1,100,000, 27 per cent. hemoglobin, and 5800 whites, the differential count showing 38 per cent. small mononuclears, 1 per cent. large mononuclear, 51 per cent. polymorphonuclear neutrophils, 8 per cent. eosinophils, 2 per cent. basophils. Microcytes were fairly abundant, as were also the macrocytes. The platelets were especially noted as being greatly reduced in number. At the present

time I think there can be but very little doubt as to the diagnosis.

We have in this patient still another phase of pernicious anemia, and one which is very well known, but frequently overlooked, namely, an onset with predominance of cord symptoms. There is no especial picture which predominates, and one may find cord symptoms readily simulating tabes or one may find such a picture as you have seen in this patient. The blood-picture, the general course of the disease, the well-marked remissions which he had last summer, the achlorhydria, the atrophy of the tongue, with the cord changes, point with unerring certainty, to my mind, to pernicious anemia. The more one studies this disease, the more one is convinced that the cord changes in many instances are present almost at the very beginning of the disease, and in some cases, as in this, dominate the entire picture throughout the patient's life. The same thing applies to the achylia or achlorhydria, which is a most constant symptom and frequently one of the very earliest. It is in my mind somewhat doubtful as to whether it is the cause of the gastro-intestinal symptoms so frequently complained of, because in so many instances, as in this, we find achlorhydria with few or absent gastro-intestinal symptoms.

**Note.**—One week later. The patient whom I showed to you at the last hour with the diagnosis of primary pernicious anemia I am unable to show you again today because he died a day or two ago, rather unexpectedly at the last. The anatomic diagnosis revealed nothing but what was expected, namely, a marked generalized anemia with no apparent cause. There were small petechial hemorrhages into the lining of the stomach, but otherwise only the customary postmortem findings in a man of his years. Sections of the cord revealed a degeneration of the lateral and posterior columns. I believe the cord is to be studied in a more detailed way.

#### SUMMARY

I think that it will be helpful if we review very briefly the essential features of the 5 cases which I have shown you.

**Case I.**—Onset twelve years ago, with great loss of weight and strength, achylia, gastric disturbances simulating carcinoma of the stomach, a blood-picture quite compatible with that diagnosis; a diagnosis of carcinoma of the stomach actually made by the attending physicians and even after consultation, the diagnosis was in doubt for some time. Diagnosis further complicated by the presence of occult blood in the stools. Patient recovered from this relapse, but the achylia persisted. Over ten years later, that is, about one year ago, he had another definite relapse, and this time with the blood-picture sufficiently characteristic to establish the diagnosis. Again a complete remission, and now the patient is in a condition of moderate relapse. I want to emphasize this type of case more especially because of its great similarity to carcinoma of the stomach. I have put on record in these clinics<sup>1</sup> a case in which a carcinoma of the stomach even more closely simulated pernicious anemia. This leads me to a further remark as to the loss of weight.

In his original description of the disease Addison called attention to the way in which the weight is preserved, and this is unquestionably true of a large number of cases. There is a tendency, however, to insist upon this as a reliable point of distinction between the Addison type of anemia and the secondary anemias, especially those due to carcinoma. I most energetically protest against this, as there are a considerable number of cases showing marked loss of weight. I have here a little analysis of 45 cases in respect to this point. Of these, 20 show marked loss of weight, so that you see that marked loss of weight, far from being rare, is very common. Losses of weight amounting to from 20 to 40 or more pounds are not infrequent. You will remember that the patient just discussed was a private patient, in good circumstances, so there was no question of poverty, neglect, or lack of competent medical care. Do not fail to remember, then, that while every severe anemia with the weight substantially unchanged speaks for a pernicious anemia, a marked loss of weight does not necessarily

<sup>1</sup> Medical Clinics of North America.

speak against it, especially in those instances in which the gastric or intestinal symptoms are marked, because from the nature of these they tend especially to produce a loss of weight and strength.

**Case II.**—The common feature of this case was the onset with stomach trouble, edema of the feet, and a marked lemon-yellow tint. A most unique feature of it was the very low point which the erythrocytes reached and which is still more striking when the completeness of the remission which ensued is taken into consideration. To anyone not accustomed to see many of these cases such an extreme remission would lead one to waver in the correctness of the original diagnosis of pernicious anemia. However, most painstaking examinations failed to reveal any cause for secondary anemia, and the subsequent relapses and remissions with typical blood findings substantiate the diagnosis. Apropos of this patient, it should be a most fundamental point in the diagnosis of pernicious anemia not to base it on the blood examination, nor the enlargement of the spleen, nor even the clinical history alone, but upon the negative outcome of a careful, painstaking, and complete examination of the individual to rule out possible sources for secondary anemia. In this connection the things to be especially emphasized are carcinoma of the stomach, bowel, and prostate, intestinal parasites, particularly the broad tapeworm, and lastly the streptococcus infections in those cases pursuing an acute course resembling the aplastic type.

**Case III.**—This patient is interesting, first, because of the extreme youth, eighteen years old at his death, and, second, because of the comparatively rapid course. I have seen one or two younger patients, but so far as I now recall they were of the aplastic type of pernicious anemia, in which the outcome is rapidly fatal and no demonstrable regenerative processes can be made out. This is the type which is simulated so closely by acute streptococcus infections. I have seen 2 or 3 cases go to autopsy with the diagnosis of pernicious anemia only to find that there was a focus of pus somewhere in the body with a generalized streptococcus infection. The large size of the

spleen in this case is not exceptional. During life repeated blood-cultures were made, and it was only after negative results from these that we ventured the diagnosis of pernicious anemia. The clear-cut history of remissions is always, as in this patient, of great value in establishing the diagnosis.

**Case IV.**—The next patient illustrated an onset with a predominance of diarrhea. It need hardly be stated that such a diarrhea would cause material loss of weight whatever the cause, but I can assure you that this patient was subjected to a vast deal of examination and laboratory work before we were content to make a diagnosis of pernicious anemia. The important thing for practitioners to bear in mind is that diarrhea may be the one symptom which attracts attention, and it may be treated over and over again with the idea that it is of dysenteric origin or due to dietetic causes without a suspicion of its being one of the expressions of pernicious anemia. I do not know whether my observation coincides with that of other clinicians, but it seems to me that these diarrheas are, temporarily at least, more easily controlled than the diarrheas due to dysenteric conditions.

**Case V.**—Lastly, we have an onset and predominance in the disease of cord symptoms, and in this patient with occasional mental symptoms, although these latter are rare in comparison with affections of the cord. It is not unusual to find numbness of the fingers and toes, and it must not be forgotten that the type of cord change is not always the same, as one may have a condition more or less simulating tabes or a spastic condition of the lower extremities, with sensory changes of various types. The important thing is that when a case presents itself with symptoms of that sort, and you have failed to find any adequate cause, to bear in mind that pernicious anemia may be at the root of the entire matter.

If now I should sum up the salient points of clinical history and diagnosis which we can derive from these patients, they would be as follows:

1. The diagnosis of pernicious anemia begins with a painstaking search for adequate causes for a secondary anemia,

especially latent carcinoma of the stomach and bowel, intestinal parasites, cryptogenetic sepsis, which is most often an acute streptococcus infection.

2. While preservation of the body fat is quite characteristic of the disease, emaciation is by no means rare, as has been commonly taught. Noteworthy loss of weight occurs in at least two-fifths of the cases, and while this suggests first of all a secondary anemia, even a great loss of weight is entirely compatible with the diagnosis of pernicious anemia.

3. A color-index of 1+ is generally found at some period of the disease, but it is often less than 1, especially during rapid remissions.

4. When the diagnosis is in doubt a clear history of a remission is a strong point in favor of pernicious anemia. In our experience distinct remissions are the rule rather than the exception.

5. An enlarged spleen is present in about one-third to one-half of the cases. The absence of enlargement does not, therefore, speak against pernicious anemia, but when other causes for an enlarged spleen can be ruled out, a distinctly palpable organ becomes a valuable diagnostic point.

6. Pernicious anemia may present itself under a number of clinical disguises, which must ever be kept in mind in the differential diagnosis.

An achlorhydria with no evident cause, stomach symptoms, or persistent diarrheas, without discernible infectious or other foundation, pallor and weakness with slight edema of the ankles, nervous symptoms pointing to a cord lesion, or a combination of these symptoms, especially when a history of one or more definite remissions of these symptoms can be obtained, point strongly to a developing pernicious anemia.

CLINIC OF DR. MILTON M. PORTIS

ST. LUKE'S HOSPITAL

**PRESENTATION OF 3 UNUSUAL CASES: I. ABSCESS OF THE KIDNEY. II. CHRONIC DIARRHEA. III. ULCER OF THE SECOND PORTION OF THE DUODENUM.**

**Case I.** Male, Aged Twenty-three, Complaining of a Sub-acute Disorder of One Month's Duration, Characterized by Weakness, Night-sweats, and Loss of Weight. Subsequent Development of a Kidney Abscess, Which Was Opened and Drained. Recovery Uneventful.

**Case II.** Female, Aged Thirty-eight, With a History of Seasonal Attacks of Diarrhea of Fifteen Years' Duration. Blood Showed Marked Agglutination for *Bacillus Dysenteriae* (Flexner). Treatment. This Case Illustrates the Value of Intensive Treatment in Chronic Diarrheas.

**Case III.** Male, Aged Twenty-four, Presents Himself Because of Severe Hemorrhage from the Stomach. Improvement Under Medical Treatment. Subsequent Recurrence. Operation Revealed an Ulcer in the Second Portion of the Duodenum. Posterior Gastro-enterostomy Without Excision of the Ulcer.

**CASE I**

THE first patient that I am to present is a young man twenty-three years old, unmarried. He complains of night-sweats, weakness, stomach trouble, and loss of weight.

**Onset and Course.**—The night-sweats were first noticed about one month ago during the hot weather. The patient did not pay any attention to these for some time, because the weather was very warm. He spent a good deal of his time



swimming and drank freely of fluids. Gradually, however, it became apparent to him that he was perspiring an excessive amount, especially at night. He had no cough and no pains in his chest. He did not think that his resistance had been lowered in any way. He had been feeling perfectly well. He had been several pounds above his accustomed weight.

Weakness developed or became apparent to the patient at about the same time. It was most marked in the morning on arising and lasted throughout the day. He continued at his work until three weeks before admission, when he was compelled to take to his bed on account of this weakness.

**Stomach Trouble.**—The patient thinks that he "caught cold in his stomach" following the night-sweats. This stomach complaint manifested itself in the left side of his abdomen, especially on taking a deep breath. The pain was located just below the costal margin. This is described as a sharp pain, but not particularly troublesome except when he takes a deep breath. This pain did not radiate and had no relation to food taking or to his bowel movements. The pain was not relieved by either of these. The patient did not notice any tenderness in this region. There was no history of nocturia or dysuria. Only once did the patient vomit. The pain lasted for two weeks and then disappeared, and he has had no recurrence during the past week.

**Loss of Weight.**—Before the onset of the present illness the patient weighed 153 pounds and was then 5 pounds over his accustomed weight. On the day of his admission to the hospital his weight was 140 pounds.

**General.**—He has had no cough and no pains in his chest. Bowels are regular, moving once or twice a day; no nocturia or dysuria; no sore throat; no shortness of breath; no swelling of feet or ankles noted.

**Past History.**—Just before the onset of the present trouble the patient had a boil in the suprapubic region which was lanced and drained, then healed completely. He was operated on for varicocele three years ago, with good result. He had the usual diseases of childhood. He had what was called influenza in

1914. There is no history of typhoid or malaria. He has never had pneumonia and denies all venereal infections.

**Habits.**—Patient does not use tobacco; uses alcohol occasionally. He leads a sedentary life.

**Family History.**—Father died at the age of fifty-three of diabetes; mother died at age of fifty-three of cancer of the stomach. He has 2 brothers, living and well, and 3 sisters, living and in good health. There is no history of tuberculosis in the family.

**Summary.**—Young adult white male is confined to bed with a subacute disorder of one month's duration which is characterized by weakness, night-sweats, and loss of weight. He has a history of pain in the left side of the abdomen which lasted for ten days and then disappeared. This developed one week after the onset of the symptoms.

**Physical Examination.**—Well-developed, well-nourished white male, twenty-three years old, acutely ill, with an anxious expression. He tosses about uncomfortably in bed.

**Head.**—Scalp is covered with very heavy dark brown hair.

**Eyes:** Pupils are regular and react normally to light and accommodation; no strabismus. Scleræ are normal.

**Ears and nose** are apparently negative.

**Mouth:** Teeth in good condition; mucous membrane of good color. Tongue protrudes in the midline and is clean. There is a small piece of tonsil in the right fossa and almost a complete tonsil in the left.

**Neck.**—Thyroid is normal. There is a palpable anterior cervical lymph-gland; no abnormal pulsation of the neck.

**Chest.**—The chest is well developed and symmetric, with good and equal expansion, but rather hyperresonant. Breath sounds are harsh; a few scattered râles heard all over the chest; no friction-rub and no increased tactile fremitus.

**Heart.**—Base is at the upper margin of the left third rib; left heart border 10 cm. from midsternal line in the fifth costal space; right heart border does not extend beyond right border of sternum. Tones are apparently clear and definite; no murmurs; no thrills.

*Abdomen.*—There is no tenderness or rigidity. Liver is normal in size and in density. Spleen cannot be palpated. Kidney is not palpable on either side. Inguinal nodes are palpable on both sides. Scar of boil is found just to the right of the midline above the pubes.

*Genitalia* are normal.

*Extremities* are well developed and no signs of trouble evident.

*Reflexes* are normal; no ankle-clonus. Babinski sign is not found.

*Rectal examination* shows no enlargement of the prostate or seminal vesicles. There is no tenderness and nothing abnormal is found on rectal examination.

Von Pirquet test is entirely negative.

At the outset repeated examinations of the urine did not show any albumin, sugar, or casts, and there were no red blood-cells found on careful examination. After a few days an occasional red blood-cell was found in the centrifuged specimen.

Blood examination showed 4,490,000 red cells; 24,000 white cells, and hemoglobin 90 per cent. Differential leukocyte count shows 16 small lymphocytes; 1 indented nucleus; 83 polymorphonuclear leukocytes. Subsequent leukocyte counts showed variations from 18,000 to 24,000, each time with a high percentage of polymorphonuclear leukocytes.

Repeated examinations of the sputum did not show any tubercle bacilli. The Widal test was negative and, likewise, the test for paratyphoid A and B was negative. Blood-culture was negative.

Examination of the stools for tubercle bacilli did not reveal any evidence of tubercle bacilli.

x-Ray examinations of the chest did not show any evidence of tuberculosis.

The temperature of the patient on admission to the hospital was 99.6° F. in the morning and rose to 101.8° F. the same evening, and dropped to 100.4° F. the following day, and then went along with an irregular curve, showing an increase in the afternoon and night as high as 103° F. on the seventh and eighth days and did not reach normal in the morning. On the thirteenth

day the temperature dropped from 102° F. to subnormal temperature of 97.6° F., and then rose again, running an irregular course above normal in the morning for one or two days, and then gradually coming down to normal in the morning. On the twenty-fifth day it remained normal morning and evening.

The pulse was slow in proportion to the temperature, being as low as 80, with a temperature of 103° F.; at other times it increased in proportion with the temperature, the tendency, however, being for a bradycardia.

Respiration rate was normal throughout.

Dr. Brawley examined the sinuses and also had an x-ray picture made, and stated that there was no evidence of sinus disease. The tonsils both showed some pathology, but there was no infection found in either.

On August 22d, which was the tenth day after the patient's admission to the hospital and the seventeenth day of the patient's illness, some tenderness was found in the right lumbar region, most marked in the region of the posterior axillary line and just below the last rib. The urine of the patient on this day showed a trace of albumin with a few leukocytes, no red blood-cells, and no bacteria. An x-ray examination was made of both kidneys and bladder to determine the presence of a perinephritic or renal lesion. The upper pole of the right kidney showed a shadow which looked like a possible abscess or a possible perinephritic abscess in this region. Dr. L. L. McArthur saw the patient in consultation, and felt that the local tenderness and x-ray evidence of the trouble warranted an operation.

This was done on the thirteenth day after the patient's admission. A small abscess was found in the upper pole of the right kidney, which was opened and drained.

An immediate drop of the temperature occurred following the operation, and the morning temperature reached normal and the evening temperature did not rise above 101° F. for the following days, and at the end of a week was normal both morning and evening. Cultures from the kidney pus showed staphylococcus. There were no tubercle bacilli found. The patient became more comfortable at once, and the blood gradually

disappeared from the urine following the operation. The usual postoperative care was carried out, including irrigations of the abscess. The patient was able to be out of bed in ten days and left the hospital two and a half weeks after operation. He gained rapidly in weight and strength and had no temperature morning or evening, and the urine again became normal.

This patient had a very puzzling picture at the outset, resembling an acute infection of the typhoid or paratyphoid type or an acute tuberculosis. There was nothing in the patient's entire picture to suggest the kidney disturbance which finally manifested itself a week after the patient's admission to the hospital. At the outset there was no tenderness to be found on deep palpation in the region which finally showed tenderness a week later. There is no doubt that the kidney abscess was secondary to the boil which was located above the pubes, and which had been incised and treated just before the onset of the patient's trouble. It is interesting that two weeks elapsed before the definite localizing findings presented themselves. It is very fortunate for the patient that the kidney abscess was the only metastatic evidence of his old trouble. From the present outlook the patient will make a complete recovery and will not have any future manifestations of his old trouble.

#### CASE II

The next patient that I wish to present was referred to me by Dr. F. H. Lamb. She is thirty-eight years old and has been suffering with a chronic diarrhea for many years. She gives the following clinical story:

Seasonal attacks of diarrhea began fifteen years ago. She could not assign any definite cause for these, but thought they were due at times to the water-supply and at other times to the fact that her food disagreed with her. The first attack lasted a few months. During the attacks occurring the following years she noticed that she was jaundiced and also was nauseated and vomited bile. There was some pain in the upper right quadrant and some tenderness in this region. The attacks gradually increased in length and in severity from year to year.

They began as early as March or April and lasted until the fall. During the fall and winter months she would be comfortable and free from diarrhea. Ten years ago during an attack she had definite gall-bladder manifestations, and a gall-bladder drainage operation was performed. She does not know whether stones were found at that time or not. Since then, although the attacks have kept up each year, she has had no jaundice.

The present attack began in July, 1921 and persisted until the time the patient was seen in November. She has had as many as nine bowel movements during the day and three during the night. There is at times marked tenesmus and often she has involuntary movements. The stools are liquid to semi-solid, with much mucus, but no blood. She has marked tympanites and passes considerable flatus. Nausea has been frequent and she often vomits; usually the vomitus is bile stained. During the periods of nausea all foods distress her. She has lost 25 pounds during the last attack. She is very nervous, excitable, and has many obsessions and fears. She does not cough and has had no night-sweats. Her gums and tongue are frequently sore, but there has been no ulceration. The gums do not bleed. Her appetite is poor. Recently she has observed some puffiness of the ankles, and this has occasionally extended up the legs during the day. This subsides during the night.

**Previous Illnesses.**—She has had the usual diseases of childhood, including whooping-cough, measles, and scarlet fever. Since then she has had only minor ailments aside from these attacks of diarrhea in the past fifteen years. She never had typhoid fever or pneumonia.

**Menstrual History.**—The menses began at sixteen. In the beginning they were regular, but with scanty flow; in the last few years they have been irregular.

**Marital History.**—She has been married four years. She has had one miscarriage of spontaneous origin. There have been no other pregnancies.

**Family History.**—One sister has attacks of diarrhea, but these have not been as pronounced or persistent as in her own

case. There is no history of tuberculosis or carcinoma in the family.

**Physical examination** reveals a poorly nourished woman of thirty-eight years with pasty colored skin. She gives evidence of having lost considerable weight; her skin is flabby and the bones are prominent.

The eye reactions are normal. There are no abnormalities found in the ears and nose. The thyroid gland is not enlarged and the cervical lymphatics are not palpable.

*Lungs*.—Expansion is normal and there are no areas of consolidation. There are some scattered râles, but no evidence of bronchial breathing.

*Heart* is normal in outline; tones are clear. Blood-pressure measures 98 systolic and 56 diastolic.

*Abdomen*.—There is a scar in the upper right quadrant the result of the previous operation. She has generalized tenderness especially along the colon, but no mass can be felt in the abdomen. The liver is normal and the spleen is not palpable.

Pelvic examination does not reveal any abnormality.

She is quite tender on rectal examination, but no mass can be felt. Sigmoidoscopic examination does not show any evidence of ulceration of the colon. A good deal of mucus was seen and marked injection was visible as far as the instrument could enter.

*Reflexes* are normal. The Von Pirquet test is negative.

Laboratory examinations of the stomach contents showed free and combined acids in normal amounts. There was no occult blood and ferments were present in normal amounts. The stomach emptied in normal time. The duodenal contents showed a normal starch reduction and trypsin was present in normal amount.

Many stools were studied, and although all of them showed undigested food and mucus and a positive test for occult blood, there was no evidence of pus or of parasites. Dr. Lamb had made very careful examinations and did not find any ova or parasites. Repeated search for tubercle bacilli was negative.

The blood at the outset showed 3,000,000 red cells, 7000



white cells, and 60 per cent. hemoglobin. The differential leukocyte count showed 66 per cent. polymorphonuclears; 26 per cent. small mononuclears; 6 per cent. large mononuclears; 1 per cent. eosinophil, and 1 per cent. basophil. The blood showed a marked agglutination for *Bacillus dysenteriae* (Flexner) in dilutions of 1:50 and 1:100, some agglutination in dilution of 1:200. The stools, however, gave no evidence of *Bacillus dysenteriae* on repeated cultures.

The x-ray examination of the heart and aorta was negative. The lungs showed an increase of the peribronchial markings, but there were no areas suggestive of tuberculosis. The stomach was hypertonic and did not show any filling defects. The duodenal bulb was normal in outline. The small bowel showed no abnormalities and was empty in fourteen hours. The appendix was found full, curved, and somewhat adherent. The cecum was likewise fixed. The transverse and distal colons were spastic and the haustral markings were not definite. There was no evidence of any filling defect or of stricture.

She was given a diet from which fruits were omitted, but vegetables, rice, starches, sugars, and proteins were given freely. She also received boiled milk in large amounts. A mixed vaccine of *Bacillus dysenteriae* was used in ascending doses in the customary manner. The colon was thoroughly irrigated daily with mild antiseptic solutions. A pineapple culture containing the Bulgarian bacillus and the *Bacillus acidophilus* were given by mouth several times a day. The number of stools reduced gradually and the liquid stools became semisolid and later formed. After a few weeks of irrigation the *Bacillus acidophilus* was implanted into the colon per rectum at intervals of several days, and later at intervals of a week. The patient is making a rapid recovery. She has gained 10 pounds in the past two months and her blood has improved. She feels stronger, has no nausea, is able to take small amounts of fresh fruit, and has semisolid to formed stools, and not more than two a day.

This patient demonstrates the value of intensive treatment in the chronic diarrheas, even when they have persisted for a number of years, as was true in this patient. It is possible

that after a time she may have some mechanical change in the colon due to scar formation, but this, I think, is unlikely. The outlook seems bright. She will have to remain on the diet and similar medication for a number of months to come.

### CASE III

The third patient that I wish to present is a young man twenty-four years old, single, who entered the hospital because of severe hemorrhage which manifested itself with bloody stools.

**Onset and Course.**—The patient has had previous attacks of this nature. He was told eight years ago that he had an ulcer. At that time he was complaining of pains which came on several hours after eating and were relieved by the taking of food, milk, or water. Later the pain began to bother him at night and in the early morning hours. At times it was so severe that it would almost double him up and interfere with his breathing. The pain is sharp, is located in the epigastrium, though at times it is felt in the back. He has intervals when he is free from pain. There has been no nausea and no vomiting. At times he has noticed that his stools are dark in color. One year ago he had definite evidence of hemorrhage in his stool. He has not followed a regular course of diet and medication in the past year except at intervals when the pain is bothering him. He does not belch or complain of sour stomach. The last attack started yesterday. He felt faint and weak, had slight nausea, and noticed a few hours later that he passed a dark stool which he recognized as one containing blood. He has had no pain in his stomach for the past few weeks. This attack evidently came on without warning.

**Previous Illnesses.**—He had the usual diseases of childhood. He had pneumonia when six months of age and an attack of pleurisy two years ago. He had frequent attacks of tonsillitis; tonsils were removed two years ago.

**Habits.**—Drinks tea and coffee moderately; uses alcohol moderately and uses tobacco freely.

**Family History.**—Father is living and well. Mother and

one brother have been treated for gastric ulcer. There is nothing else of significance in his family history.

**Physical Examination.**—The patient is robust and well built. He entered the hospital on a stretcher and has been kept in bed quiet since entry because of his hemorrhage.

Examination of his eyes does not reveal any abnormalities; the ears and nose are negative. The teeth are in good condition. The tonsillar fossæ show no evidence of a return of the enucleated tonsils. The cervical lymphatics are not palpable. The thyroid is normal in size. The lung findings are normal. The heart is a little rapid, but its size and sounds are normal. There was no attempt made at palpation of the abdomen because of the bleeding. The genitalia are normal. The reflexes are normal and there is no evidence of involvement of his nervous system.

The blood examination does not show any marked anemia. The hemoglobin is 90; red blood-cells 4,600,000; white blood-cells 8700. Coagulation time is four and a half minutes.

The urine shows negative findings on repeated examinations.

The stools show the typical tarry, bloody stool of hemorrhage.

The Wassermann test was clearly negative. No attempt was made at examination of his stomach contents or x-ray examination of the gastro-intestinal tract because of the hemorrhage.

He was given nothing at all by mouth. An ice-coil was placed upon his abdomen and hypodermics of morphin were given in sufficient amount to keep him quiet and to control the desire for bowel movements. Small amounts of water containing glucose were administered by the bowel. The appearance of the stools after one week was such as to indicate an absence of blood, but occult blood was found for several days longer. After three days the patient was started on a milk and cream diet, getting a small amount every two hours, followed by calcium carbonate with bismuth subcarbonate. Inasmuch as the bleeding was not increased, this type of food was rapidly increased in amount, and after a few days ice-cold milk and

cream in 8-ounce doses were given every two hours, and this was followed by the powder mentioned. The bleeding did not recur. The patient was very comfortable, and from this stage the diet was increased, using puréed vegetables, eggs, soup, and fruits, along with the milk and cream mentioned. Feedings were kept up every two hours and a sufficient amount of alkali given as is customary in ulcer cases. Cereals were later added to the diet, and after four weeks the patient was allowed to sit up in bed and a week later allowed to go home. During all of this time no stomach tests or x-ray examinations were carried out, for it seemed that the diagnosis of duodenal ulcer was clearly shown by the previous history of the individual and the present picture. It was my plan to have the patient go through an x-ray examination a few weeks later when his condition would permit.

During the time he was waiting for this subsequent examination he visited some friends in Canada, and while there had a sudden severe hemorrhage. He was given the same sort of treatment in Canada which he had been given here. An attempt was made at an x-ray examination, but the doctor found nothing definite. On his return to Chicago he consulted Dr. L. L. McArthur, who advised operation because of the recurrence of the bleeding in spite of the prolonged and thorough ulcer management.

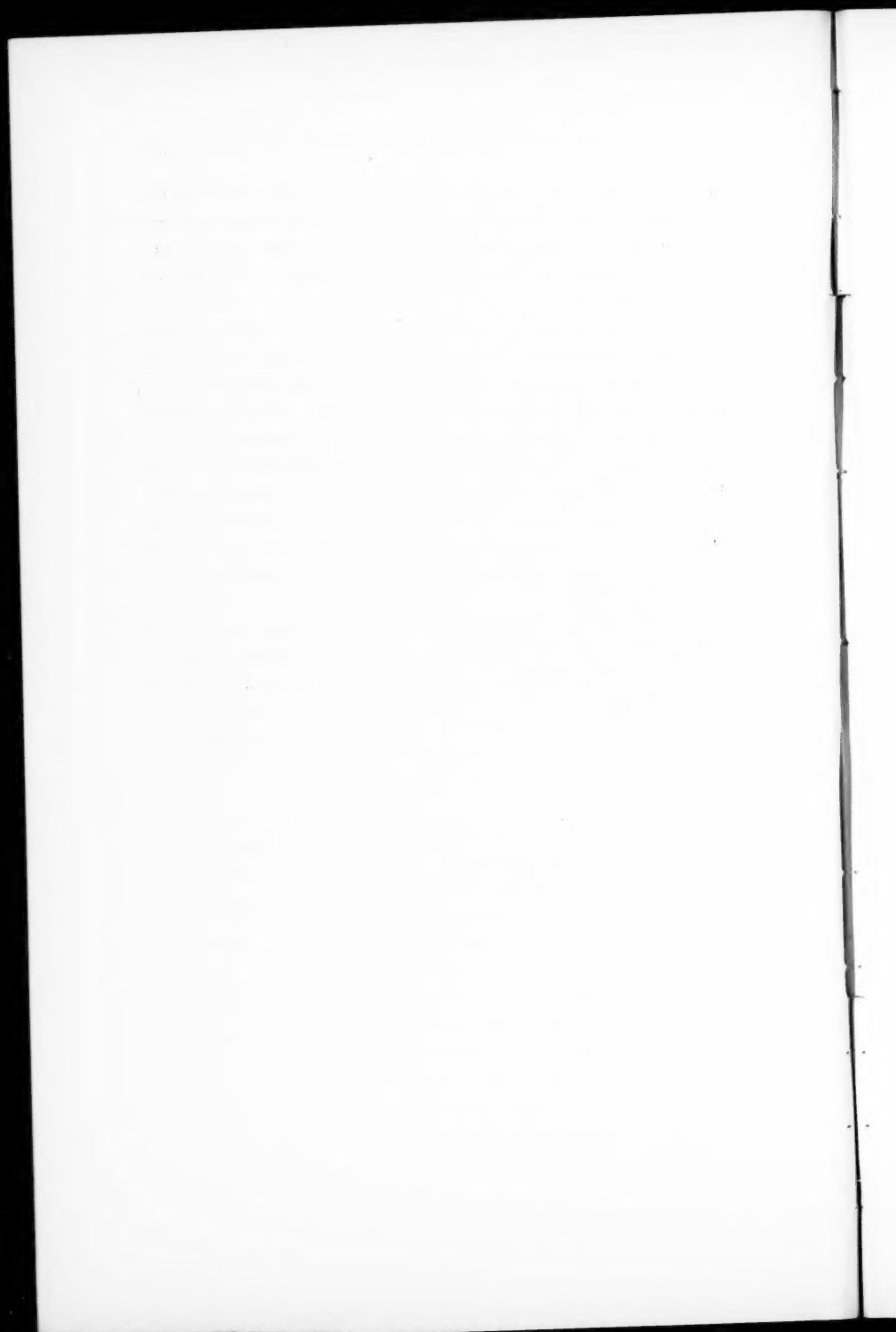
He was operated upon, and there was no evidence of ulcer of the stomach or of the first portion of the duodenum seen. On thorough examination Dr. McArthur discovered an induration in the second portion of the duodenum lying posteriorly. It had a deep crater and penetrated the pancreas. Because of this the ulcer was not excised and a typical posterior gastro-enterostomy was done. Since that time the patient has been following a diet and medication which will insure healing of the ulcer. With his gastro-enterostomy there is a more definite hope of success in avoiding future hemorrhages.

According to Carman nine-tenths of the ulcers are seen in the first 2 inches of the duodenum, and these are usually on the anterior wall. The remaining one-tenth may be found

in any part of the duodenum. However, ulcers beyond the first portion are distinctly uncommon. Posterior ulcers are very prone to perforate and invade the pancreas or liver and produce auxiliary pockets. This makes surgical excision extremely difficult.

It is interesting in this case to note that the x-ray work and the examinations at operation both seemed negative for ulcer, and it was only after very careful examination of the lower duodenum that the exact nature of the pathology present was revealed. It is also worthy of comment that the mother and brother of this patient suffered from similar trouble, and it makes one reflect on the possibility of some type of streptococcus infection in the family, probably associated with tonsillar disease. I did not have an opportunity of observing the other two members of the family and can add nothing in detail concerning them.

I shall watch with great interest the further progress of this patient and will have later x-ray examinations made to see if any further evidence can be produced to show the progress of the healing.



## CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

### TWO CASES OF HYSTERIA

**Two Cases Illustrating a Phase of Hysteria Not Frequently Spoken of. Discussion of Symptoms and Physical Signs in Each Case. Treatment Employed. Result.**

It has been said that hysteria can simulate any disease, that it can imitate any of the symptoms of disease. There is a phase of hysteria that is not frequently spoken of, namely, the tendency for symptoms that may have had some organic basis to persist after the organic basis has disappeared.

In the discussion of the causes of hysteric or functional symptoms the sense of inferiority has been much emphasized, especially here in America. This is perhaps because competition plays a greater rôle with us than in the older countries. The absence of fixed strata in the American social structure encourages competition. Success has the inevitable corollary of failure for the opponent. If one group of men succeed, their success is directly or indirectly pictured, made manifest and spectacular by their superiority over some other group of men, which is equivalent to saying the second group are made to feel their inferiority to the first group.

With individuals it is as with groups. Every little human animal sees itself the center of the universe: a child is entirely self-centered; this is as true back of the Yards as on the "Gold Coast." One of the most important functions of education is to teach the egregiousness of such a point of view, and a mark of normality is the ability to accept with an outward show of equanimity the evidences of one's limitations.



Now, the limitations may be mental or social, or they may be physical. To put it crudely, John and Bill get into a situation in which John fails because of manifest mental inferiority, or Mike's social position leads to his failure, or Jim's smallness of stature is a deciding factor; John's attention may be directed to the palpitations of his heart and so he develops a hypochondriasis; Mike gets drunk and Jim grits his teeth, digs in, and makes a mental giant of himself. Of the physical limitations there are deformities and other inadequacies, and there are organic diseases. It is with these last that I wish to deal here.

The first case that I want to describe to you had been seen by men whose clinical observation is of such a high grade and the evidences of disease had been so unmistakable that no one could deny that the patient had had some organic lesion in the brain, probably in the brain stem. However, when I first saw her, a year after the onset of the trouble, there was no denying the functional nature of practically all of the signs and symptoms presented.

#### CASE I

Miss X., aged twenty-seven years, was first seen on January 17, 1922. Her history, as written by the intern and resident physician, was, in brief, as follows:

At twelve years of age a tooth was crowned and from then on she had headaches at irregular intervals. They were not "sick" or menstrual, but were quite frequent. In 1920, at the age of twenty-five, because of a slight thyroid enlargement, she was sent to a large surgical clinic where her basal metabolism rate on two occasions was +9 and +10, an excess practically negligible. Her chief symptoms were tachycardia, her pulse running up to 140 per minute, and nervousness. She returned home with a diagnosis of slight hyperthyroidism and infected (septic) teeth. Upon her return home the teeth suspected were drawn and the tachycardia and headaches disappeared. For a year she felt very well, during which time she worked hard as a saleswoman. A year ago her teeth were x-rayed. On January 17, 1921 two more teeth were extracted; one of them, the only crowned tooth, was the one crowned at the age

of twelve. This was a lower molar, which showed a large abscess at the root and some bone necrosis. The cavity was drained. Thirty-six hours later she was awakened by a chill. She had a temperature of 103° F., her pulse was rapid, and her doctor found a systolic murmur over the mitral region and a tumultuous heart action. Her mouth was "in fearful condition." The fever continued for four weeks. In spite of the serious nature of the onset, she remained up and around, going to the dentist for treatment. About two weeks after the teeth were removed, while ironing, she suddenly had a feeling of fulness in the back of the head, "as though she might have a stroke." There were also double vision, vertigo, and "neuritis pains" in the arms. She was then put to bed. About one month later she had five days of terrible headache, as though "someone was hitting her with a hammer 3 inches above and behind the left ear." She had double vision for a day, the temperature was subnormal, the pulse 56, the blood-pressure 110/160, frequent vomiting was present, and the reflexes were increased on the right side of the body. After five days of pain in the head she had a hemorrhage from the left nostril, with some relief from the headache. (I think it is well to bear in mind the fact that these details are given by the patient herself.) Several days later there was a deviation of the tongue to the right and a partial paralysis of the right side of the face, and on walking she always seemed to fall to the left. The left pupil was considered larger than the right.

These episodes occurred in January and February, 1921. A low fever (99° to 100° F.) persisted throughout the summer. On March 26, 1921 the left antrum was opened and washed out through the nostril. At the end of April a left upper tooth was extracted, exposing the antrum, and irrigation was continued through the sinus. In June she went out for a short motor ride, but the headache described above and behind the left ear returned, and she had to be carried into the house. She vomited several times and wished that she could die. She has been in bed since June with headache, weakness, fever, and other symptoms which will be described. Throughout the

summer she had hay-fever. In July an endocarditis was diagnosed by a very competent internist. About the end of August her headaches became much worse, her tongue was thick, and her speech could scarcely be understood. Early in November her headache suddenly became greatly aggravated over night, diplopia returned, and the head was retracted for three weeks. Late in November she began having severe pains in both sciatic nerves. They were always tender. This pain had been getting more severe and the tenderness increasing, so that for the four weeks before entrance into the hospital, January 16, 1922, she had been unable to straighten her legs. In fact, the sciatic pain was so severe that for a long period she had received 20 grains of bromid every four hours with  $\frac{1}{8}$  grain of morphin by mouth, and once or twice a day  $\frac{1}{4}$  grain of morphin hypodermically.

Repeated Wassermann tests were negative. There was no middle-ear disease at any time. Frequent white blood-counts never showed more than 10,500.

The family history was negative; there was no tuberculosis. The patient had worked rather hard before her illness and seemed to be an entirely normal person, taking a keen interest both in her work and in her pleasures. Her menstrual periods were normal. She had had measles, scarlet fever, and mumps in childhood; hay-fever had made her summers difficult since the early age of seven years. Her tonsils had been removed in 1912 and the frequently recurring sore throats had ceased.

Examination by the intern upon admission showed a thin, anemic woman, acutely ill. She was rational; there was some eruption on the face and front of the chest. A great many things were absent as to the scalp, the face, the ears, the nose, and the eyes. It was stated, however, that she had a diplopia. There were many carious teeth and considerable pyorrhea. The tongue deviated to the right. There was no thyroid enlargement; the lymph-glands in the left cervical triangles were slightly enlarged. The breasts were small, firm, and no masses could be palpated. The chest and lungs were normal. The heart outlines were the right sternal border, the fourth inter-

space, and well within the left middle clavicular line. There were no murmurs or thrills. It was stated that there were two good tones at the apex, that the first tone in the aorta was normal, the second accentuated. The abdomen was normal. The lower extremities were described as atrophied, with marked pain on passive movements; the jaw-jerk was normal; wrist-, triceps-, knee-, and ankle-jerks exaggerated, with exaggerated bilateral ankle-clonus. No Babinski sign was elicited.

A competent ophthalmologist pronounced the eye-grounds normal.

From the foregoing history one could very well diagnose organic disease of the brain. The severe pain coming on abruptly soon after stirring up an old focal infection; the tooth extraction, the diplopia, dizziness, vomiting, and the heart symptoms might all have been due to a focal disease in the brain stem or a basal encephalitis of the epidemic type. You know that a large number of the typical cases of epidemic encephalitis had much that history—some pain at onset, diplopia, and temperature running 100° to 102° F.

Let us take up each symptom and consider it as to whether it speaks for or against organic disease.

There are two varieties of pains spoken of, the parieto-occipital headache and the "neuritis" pains in the arms. When epidemic encephalitis began with severe pain it was highly characteristic to have it described as being in the face, the neck, the arms, the trunk, or the leg. This subjective symptom, then, may have evidenced encephalitis.

The diplopia is difficult to evaluate. If the doctor had described it as a strabismus, an objective sign, rather than as a diplopia, a subjective one, it would have meant much more. When I examined the patient and inquired into the diplopia in detail she gave it this curious description: "I'd see the doctor coming into the room twice (meaning as two figures), only the second one was much smaller than the first," and one of the two figures was described as "dim and hazy."

The vertigo was described as a sensation of constantly swinging on a rope counterclockwise. This is a description

of the symptom at the time of examination. It is perhaps useless to discuss it, although it does show somewhat as to the sort of person we have to deal with. When a patient makes a symptom picturesque it is safe to assume that this quality is desired by the patient: That the patient wishes the world to be fully aware of the nature or severity of the symptom. This quality arouses a suspicion. I think it justifies the assumption that the patient gets some pleasure out of the possession and description of the symptom.

There seems to be little doubt that the patient had fever over a long period of time, but here also there was something peculiar. When she entered the hospital the patient was taking her own temperature per axilla. There seemed to be no satisfactory reason for this. It was discontinued and rectal temperatures were taken. The rectal temperature showed distinct variations, running from 100.2° to 98° F. (Fig. 289).

Now as to the tongue protruding to the right. It certainly did when I examined her, and it had all the appearance of a tongue deviating toward the paralyzed side. She could not protrude the tongue from the left side of the mouth, but she could, and did, exert very strong pressure against the inside of her left cheek with her tongue.

Now this, of course, is an obvious inconsistency. She appears to be unable to protrude the tongue in the midline because of weakness of the right half, she is unable to protrude the tongue from the left side of the mouth because of weakness of the right half, but she can force her tongue against her left cheek with very good strength, strength of the right half of the tongue. Any one of you put the tip of your tongue against your left cheek, gently put your finger on your tongue and force your tongue against your cheek, and you will feel the right half of the tongue grow quite hard, as should a contracting muscle.

This inconsistency is of the variety that one sees in the functional patient who has been frequently examined and whose symptoms may have been commented upon or discussed before her. You know that the stereotyped criterion of paralysis of the tongue is that it protrudes from the paralyzed side of the

mouth and that it protrudes toward the paralyzed side. Nothing is said about an inability to force it against the opposite cheek, and when, as in this case, one sees a tongue deviating as does this one, but which can be forced against the opposite cheek, you are privileged to put your own tongue in your cheek.

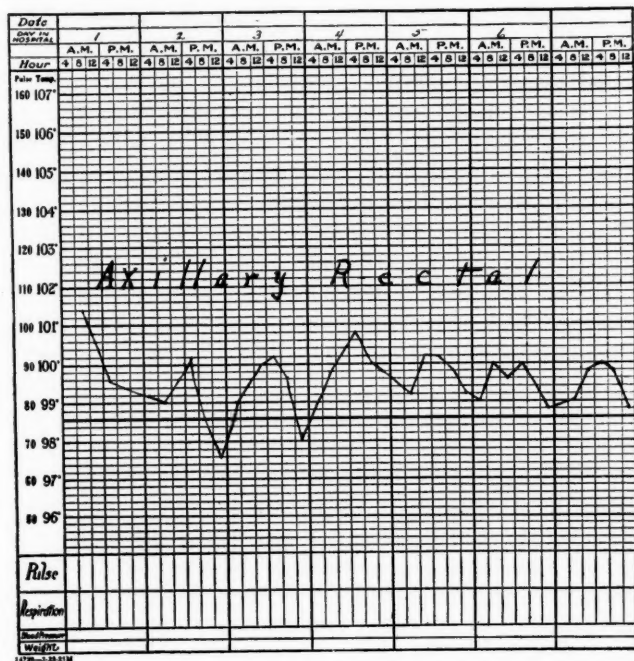


Fig. 289.—Case I. First three days temperature was axillary, the last four, rectal. The axillary curve has a higher maximum and has greater daily variations. As a usual thing rectal temperature is 1 to 1½ degree higher than axillary.

There was an element in this sign that struck me as particularly significant of the functional, and that was in the way she sought to protrude her tongue from the left half of the mouth. Her tongue moved around in the left half of the mouth as if seeking the opening, but unable to find it. All in all, it

was a perfect demonstration of a patient trying to produce the sign or symptom in a classical manner, in a manner that would appeal to the medical man.

A partial paralysis of the right half of the face, pupillary inequality, and exaggeration of the reflexes on the right side of the body may all have been present and due to an organic lesion, a lesion of a temporary, inflammatory nature. They were described as appearing in the period of illness in which there were generalized symptoms of infection following removal of an infected tooth, namely, chill, temperature of 103° F., rapid pulse, tumultuous heart action, and a mitral systolic murmur, with a blood-pressure of 160/110. I can say nothing as to the significance of this last sign.

When I first saw the patient practically all these symptoms had disappeared or been replaced.

The face did have an asymmetric look, but that was discounted by the very bad condition of her mouth. The patient had been taking rather large doses of bromids and eating almost no solid food. Her lips were badly cracked, so, naturally, movements of the face were interfered with.

The reflexes were greatly exaggerated, but this was as true of one side as the other; it was not hemilateral. The temperature curve was curious. As I have said, the patient was taking her own temperature by axilla and, as you see (indicating chart) (Fig. 289, Case I), it ran from 101.4° F. on admission to the hospital down to 97.4° F. in forty hours, a much greater range than was shown by the rectal readings supervised by the nurse. I think one is justified in saying that such a curve is quite without value, except that it excites suspicion in the medical attendant's mind—a suspicion that the record is not genuine.

When the patient was first seen her legs were drawn up under her "because of the sciatic pain." In attempting to elicit the ankle and knee reflexes I drew her legs out, and while talking about reflexes manipulated the legs rather roughly. After seeing the marked clonus in knees and ankles I said that the sciatic pains must be much less to allow of such a marked clonus. I was then invited to look at her back, where there



was a rather marked discoloration of the skin over the sacral region, such as one sometimes sees as a result of long-continued application of heat. I was properly impressed with this, but explained it volubly, as the sort of thing known to occur under such circumstances. While talking I pressed hard over this whole region without appearing to pay any attention to complaints of pain, reassuring her as I did so on the unimportance of these symptoms.

Then there was the clonus. It was well sustained, rapid, and forceful, but it was very irregular. At times some of the downward thrusts of the feet were much more vigorous than others. Also in the test for ankle-clonus one could feel the thigh muscles come into action of a clonic nature, reinforcing the muscles of the leg.

The knee reflexes were greatly exaggerated. In fact, a tap on the patellar tendon set up a clonus, but once when I hit the tibia well below the insertion of the tendon there was also a well-marked reflex, and again I started to strike the tendon, but stopped the hammer before it struck, and there was a very brisk response.

Now, of course, these last responses could mean just one thing, and that was that the patient was trying to make the most of her symptoms, was trying, for some reason all her own, to impress the examiner with the degree of her disability. When viewed in this light the signs all take on a slightly different appearance. Certainly one is forced to be skeptical concerning all her subjective complaints of pain, headache, diplopia, dizziness, and weakness.

There were two other elements that help to complete the picture. She says that when she was lying on a cot on the train coming to Chicago something suddenly snapped in her head, and since then she has been much better as to pain in the neck, retraction of the head, and headache. Now I know of nothing physical that could be invoked in explanation of such a phenomenon. Certainly no abscess is going to break, though the description of the occurrence made me think of the nose-bleed that had previously relieved her somewhat. Also, no

vertebra is going to slip back into position. The only explanation possible is that the symptoms are better because of an improvement in the functional state.

The second element is this: I had reassured her. I then went to the hospital drug room and came back with some capsules, an eye-dropper, and a bottle of fluidextract of cannabis indica. Then, very earnestly, I instructed her mother just how to drop 5 minims into a capsule and give it to her just before each meal, going on to say that when she had had a certain amount of this prescription electricity and massage would be enabled to affect her in an efficacious way. As I think I have said, the patient was getting morphin sulphate, grain  $\frac{1}{2}$  hypodermically, and other medicines every day. From the beginning of the cannabis indica treatment all this medication was stopped and no request was made by the patient for its resumption during the following two and a half weeks of her stay in the hospital. With the cessation of the bromid and morphin and with the patient's beginning to eat again, the very bad condition of the mouth and tongue cleaned up rapidly.

The day after my first examination the patient volunteered that her diplopia had disappeared, and with it the headache, dizziness, and sciatic pain. Before she left the hospital she was protruding her tongue normally.

Now, of course, this is nothing surprising. When hysteric patients get ready to drop their symptoms they do so—that's all. And it does not matter what means are employed or what bunkum is used. However, when a patient has been in bed and disabled for a year some allowance must be made for her. The examiner must seem to fall in with the patient's idea if he wishes his therapy to prevail. He cannot expect anything from trying to show them up to themselves, their family, and friends as impostors. Hence, the necessity for the careful examination and the prescription. So far as I know I might just as well have prescribed chalk, bread, or beeswax as cannabis indica. I do not know that the cannabis indica is really active. However, it does sometimes give the susceptible a sense of swimming in the head.

This case is cited not because of the efficacy of the medicine, but because of the fact that there seemed to be a functional, or hysteric, persistence of symptoms which originally might very well have been organic. The proper way to have gone into the case would have been by means of psycho-analysis, but efforts in that direction, made tentatively when I had satisfied myself of the functional nature of the symptoms, were absolutely unavailing. Hence, the bunkum.

In conclusion, it is fair to say that there is much danger in being guided by the general attitude of the patient. However, in this case the disingenuousness of the woman was too striking to be overlooked. It appeared in the temperature curve, in the disability of the tongue, and in the knee- and ankle-clonus. These so-called subjective signs were not genuine; hence, they made one doubt the genuineness of the subjective complaints—the pains, dizziness, diplopia, etc.

#### CASE II

The second case may have some organic disturbance, such as the inclusion of a small branch of a sensory nerve in a scar, as the legitimate background for the main symptom, namely, pain in the lower jaw. There is, however, or at least there was, an analgesia over the side of the face on which the pain was felt, which was in large measure, if not entirely, hysteric or functional.

This woman is sixty years of age. She is big and strong, florid of face, and looks the picture of health. There is nothing in the general examination to indicate her years. The blood-pressure, heart, urine, etc., are all normal. Nineteen years ago she had 26 teeth extracted at one time because she had had a great deal of trouble with toothache, and several of the extracted teeth were abscessed. Plates were adjusted in due time and she was free from pain for about ten years. Then one evening—and I am here giving the story in her own words—she made piccalilli, and the next morning there was numbness in the left half of the mouth and cheek, and she began at the same time to have pain in the left side of the face. It was burning

and situated somewhat deep in the tissues, running beneath the lower jaw bone.

About five years ago she was operated upon and a piece of "dead bone" was removed from the left lower jaw. Since then the "drawing sensation" in the face has not been so severe.

Up to the time of my examination she had been having the pain more or less constantly. At times, namely, when she was exposed to cold wind, or when she rubbed her lower jaw about an inch to the left of the chin, the pain would be more severe. At no time has she had any trouble in the tongue. Upon questioning she allows that there are times when she has a rather sharp pain shooting down the lower jaw.

The pain never keeps her awake and never wakes her from her sleep. Eating and drinking seem to have no causal relation to the pain.

So far the history sounds as though there were some organic cause for the pain, except that it is hard to believe that such a healthy appearing woman is constantly suffering and has been suffering a steady pain for nine years.

Upon examination the most striking feature was the analgesia. I could jab her in the face with a pin anywhere on the left cheek and she would describe each jab as "dull." The outlines of this area of analgesia were peculiar. She was wearing spectacles and the upper line certainly was determined by the bow of the spectacle frame. Jabs with a pin up to the bow were described as "dull," but above the bow were winced from and called "sharp." The lower line coincided with the line of the lower border of the inferior maxilla. The analgesia did not run to the middle line, but stopped at the border of the nose and upper lip, and ran to about 1 cm. of the middle line of the chin, in this way (demonstrating on chart).

Such an area of analgesia fails to fit in with what we know about either segmental or peripheral nerve distribution. This is in the area supplied by the fifth nerve. There is an overlapping of the superficial cervical nerves which extends about an inch up on to the cheek from below.

I think it is safe to say also that if the analgesia was purely organic in origin it would have run closer to the middle line of the chin and would have included most of the left half of the upper lip and the left side of the nose. It is conceivable that the upper border was determined by the distribution of the middle branch of the fifth nerve, the upper, or first branch, supplying the forehead, having escaped. However, this line followed the bow of the spectacles so exactly that it was impossible to overlook the coincidence.

The patient was first seen two months ago. In the interval faradism had diminished or done away with a large proportion of the analgesia, so that now (March, 1922) there is merely a small area about the size of a silver quarter just below the angle of the mouth. At one time even this area seemed to possess normal sensation.

When I first began treatment the pain was relieved for periods of one-half hour to two hours following the faradism. Now she usually has relief for five or six days, but even after this long period of time it seems impossible to get complete relief.

One is tempted to make a straight diagnosis of hysteria. It is impossible to elicit any of the mental stigmata of hysteria unless we can consider that removal of the analgesia constitutes such. It is such pain as this that is frequently spoken of not only by the patients, but by their medical attendants, as *tic douloureux*, or facial neuralgia, and then is treated with alcohol injections.

It might be well to inject the inferior branch at the mental foramen in this case. It would certainly produce a considerable psychic effect, and so might be the right thing to do. However, the paresthesia following upon an alcohol injection is frequently very troublesome, and one hesitates before taking the chance of starting something that one cannot see the end of.

This condition might easily have been diagnosed trifacial neuralgia, but, fortunately, it was recognized by an oral surgeon as being something different. It is of sufficient importance to point out the pathognomonic character of the pain in trifacial neuralgia because so many pains in the head and face

are diagnosed as trifacial, and are treated as such with injections into the gums, the skin, the nerves—in fact, almost anywhere.

The pain of trifacial neuralgia in the early stages is always flash-like or stabbing and never continuous. If it is described as continuous for an hour or more it is not *tic douloureux*. That is a point made clear and insisted upon by Patrick. After a long continuation of such flash-like attacks—each pain may last up to three or four minutes, although usually they are only one-half to three-fourths of a minute in duration—after months or years of attacks not infrequently the patient will complain that in between the flashes there is a dull discomfort that is more or less continuous. When the patients, uncoached and unquestioned, say they have a severe pain in the face that lasts for several hours, the safe guess is that it is not *tic douloureux*.

Now this patient's pain is continuous and has been so for nine years, and still she looks the picture of health and contentment. Either her complaint is exaggerated in the telling or else she has a marvelous ability to stand pain.

To bear out the diagnosis ruling out *tic douloureux*, it is impossible to start pain by manipulation of the skin or mucosa of the left half of the face and mouth.

I think we can safely say that this is not a case of *tic douloureux*, but what is it? In the first place, the mode of onset sounds suspicious to me. Such a sudden onset coming on during the night and not waking the patient is difficult to explain. Also for the patient to have remained practically impervious to time and treatment seems strange. If due to anything serious it seems as though it should have shown some decided external signs by now, either that or have improved greatly. But the really characteristic element in the picture is the outline of the "numbness." She bore the pricking of the pin up to the line of the bow of the spectacles, where the bow crossed her temple, but on the upper side of the bow there was normal feeling. The analgesia persisted to the lower border of the lower jaw, from angle to chin. As to the upper line of the analgesic

area, it was very decided, and was determined, as I have said, by the bow of the glasses (Fig. 290). One might think that this was determined by the line of separation between the supra-

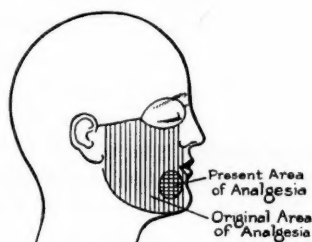


Fig. 290.—Case II.

orbital branch and the middle and lower, the infra-orbital and inferior maxillary branches, the first being normal and the second and third being analgesic. However, sharp lines of de-

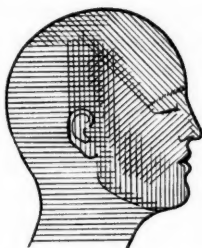


Fig. 291.—Case II. Sensory supply of head, face, and neck.

- ≡≡≡ Branches of the cervical plexus.
- /// First or supra-orbital branch of trigeminal or fifth nerve.
- |||| Second or infra-orbital branch of fifth nerve.
- ||||| Third or inferior maxillary branch of fifth nerve.

marcation between the three branches are not common, and it is usually quite difficult to distinctly outline the superior branch after injecting the middle or inferior, or both. There



is a well-marked overlapping, as is shown in this chart (indicating Fig. 291).

But still further from the expected is the lower line of the sensory loss. As you see, there is marked overlapping here also; the branches of the superficial cervical nerves course up over the lower border, spread out over the lower portion of the cheek, and reach an inch or more above the line shown by this patient.

The lower line of analgesia is what is regularly seen in the functional cases: it conforms to a topographic line, not to a physiologic line, just as we have a loss of sensation of the leg just to the knee or groin, or of the arm just to the elbow or shoulder. Also, the analgesia has diminished very quickly, considering it is supposed to have lasted for several years, under faradism. Whereas it formerly involved the whole cheek, it now is reduced to an area about the size of a 25-cent piece just below the left angle of the mouth (see Fig. 290). There is nothing in faradism which will of itself produce such a result; the psychic effect of faradism must be held accountable. The analgesia must have been functional in the area from which it has receded.

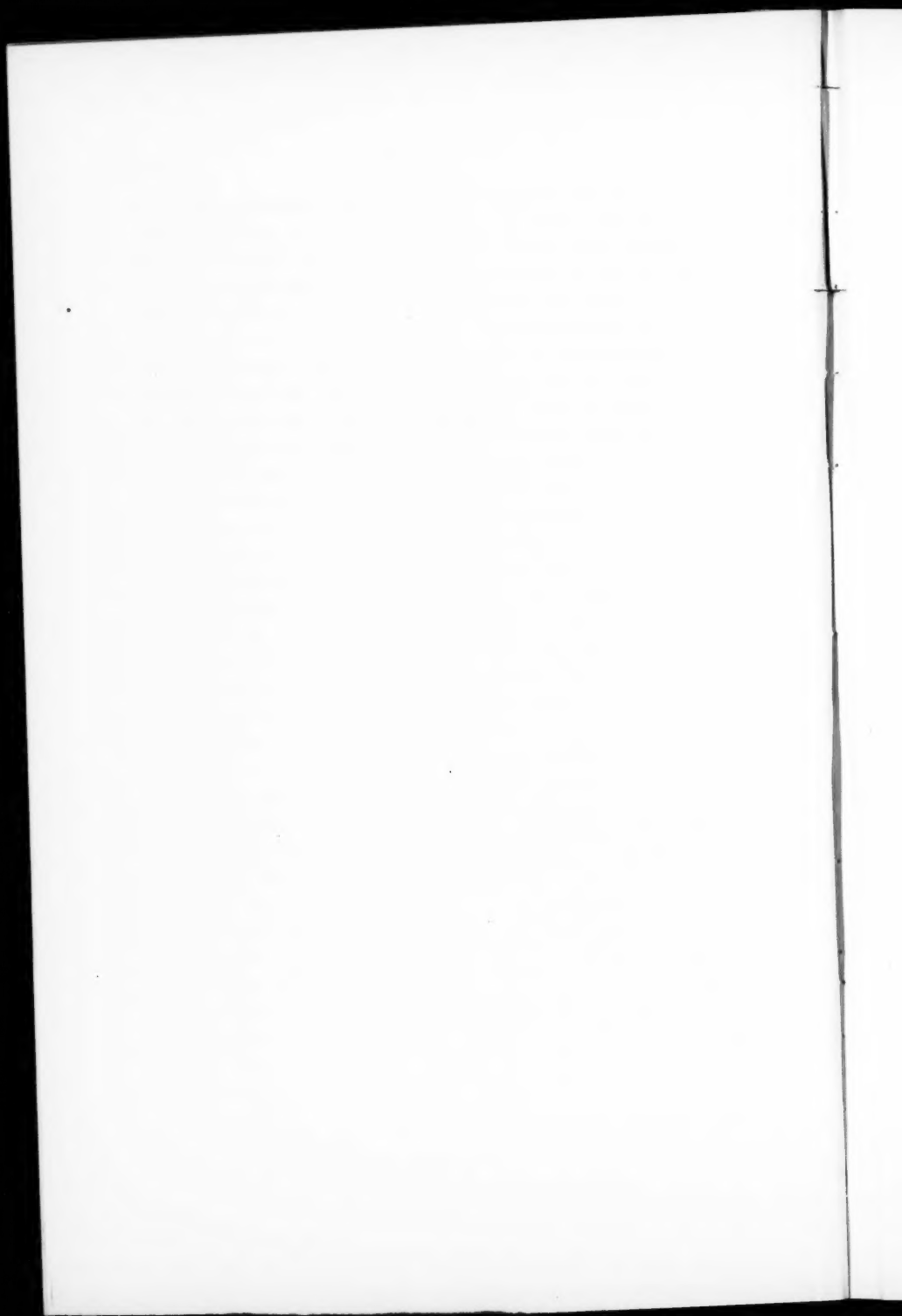
I am not sure as to the small area remaining. It may be due to the operation described by the patient, in which a piece of the inferior maxilla was removed. In that operation a small branch of the inferior maxillary nerve running to this region may have been destroyed and a permanent analgesia established.

This may be true, but I am rather inclined to believe that this analgesia also is functional. In the first place, it does not run to the middle line as I would expect it to; in the second place, I am almost certain that it also disappeared for a short time and reappeared when I, unavoidably, failed to keep an appointment with the patient. In the third place, when some of the analgesia is so clearly functional, I am inclined to believe it all is until I am proven wrong.

Now these 2 cases have in common this fact: in both there has been physical disease which has largely cleared up, and in

both symptoms similar to those of the organic disease have replaced the original symptoms. In the first case these new symptoms were quite different, very much exaggerated, and quite severe. In the second case there was a functional analgesia in that side of the face from which a piece of dead bone was removed five years ago.

**Concluding Note.**—This patient was seen on May 8th. She stated that she had been free from pain since the last treatment with faradism; one month had elapsed. The small area of analgesia below the left angle of the mouth still persisted.



## CLINIC OF DR. CLIFFORD G. GRULEE

PRESBYTERIAN HOSPITAL

### INFANTILE ECZEMA

**History of Case Presented. Theories Regarding the Cause of Eczema in Children. Treatment Employed in Present Case. Advantage of Hospital Treatment in These Cases.**

I WANT to show you today this baby boy, aged seven months, who came into the hospital on February 2d. His physician called me up the day before his appearance here and stated that the child had eczema for four months which he had not been able to influence in any way. The history as obtained by the intern was as follows:

The eczema began five months ago with a rash on the cheeks, then spread to the forehead, ears, neck, shoulders, and body. It reached its worst stage two months ago, since when it has been alternately better and worse. There have been times when the scalp was a solid mass of scabs and, again, it would be perfectly clean. Only part of the time was the surface moist and weeping. The infant is always trying to scratch, and will do so until the skin bleeds if permitted.

The past history is as follows: He was a breast-fed baby for three months, being nursed every three hours—six feedings in twenty-four hours, and no feeding between 7 P. M. and 4 A. M. The mother apparently had a large amount of rich milk. After the eczema appeared an attempt was made to dilute the breast milk in the diet by giving the baby lime-water and barley-water, 2 ounces before each nursing. As there was no improvement after a month, the child was weaned entirely and put on modified whole cow's milk with lime-water. Then, as no im-

provement took place, a succession of changes were made and several combinations tried, but all this time an attempt was made to underfeed the baby. Various patented foods together with cow's milk have been tried, but without success. The last food was one rich in condensed milk and malt food.

On Friday, January 27th, a rather severe diarrhea started. The stools were almost constant, colorless, and watery. On Saturday the child was better, but up until Wednesday, February 1st, there was no very great change. The child on entrance seemed to be improving. It had vomited everything on Saturday and Sunday, but ceased this by Monday.

There is very little in the birth history. The child was a full-term, normal baby, and reputed to have weighed 10 pounds. He gained up to 14 pounds by the end of the third month, but has lost since.

**Family History.**—Parents are living and well; this is the only child. There have been no miscarriages. There have been cases of localized eczema in the adults of both sides of the family.

**On examination** we found the child restless. He was covered with eczema. This was of the scaly variety, with crusts on the face and exposed portions. There was not a square inch of the body that was not covered by a thick eczematous process, and the scalp was crusted thick with heavy crusts. The child was miserable, making every attempt possible to scratch itself. It was very evident from the condition of the skin that he had lost weight rapidly. The skin was in folds and there was a loss of tissue turgor. The elasticity was very much reduced.

Physical examination of the child revealed little else than the presence of many lymphatic glands in various regions of the body, evidently secondary to the infectious process in the skin. On entrance the weight was 9 pounds, 12 ounces. The temperature was 97.2° F., but it rapidly rose that evening to 103° F.

The blood showed 4,840,000 red cells, 10,400 white cells, and 90 per cent. hemoglobin. Of the white cells, 40 per cent. were small lymphocytes and 3 per cent. large lymphocytes;

4 per cent. were large mononuclears, 40 per cent. polymorphonuclears, and 10 per cent. eosinophils. The urine was negative.

Since entrance the child has shown a rapid gain in weight, the weight now being 10 pounds, 5 ounces, a gain of 9 ounces in six days. The temperature, however, has varied, the rises being usually as high as 102° F., though there seems to be a tendency toward a decrease in temperature today.

We have here, then, a baby seven months old with a very marked eczema which has existed for five months. This has been present in spite of repeated attempts to overcome it by change of food and various ointments. The most interesting thing about the eczema is that it persists in very severe form and degree, although the child showed a very definite evidence on entrance of having lost rapidly in weight, and the history is that of a recent diarrhea with vomiting. The condition of the skin aside from the eczema is of great importance to us in this instance. The history of diarrhea, while definite, gives us no idea of the amount of loss of weight, but the loss of tissue turgor is such as to assure us that the loss of weight has been very marked in a short time. The interesting thing about the blood-picture is the presence of 10 per cent. eosinophils in the white blood-count. Eosinophilia is quite commonly present in severe cases of eczema.

The one feature about the case which is rather hard to explain is the temperature. Although since his presence in the hospital there has been no diarrhea or vomiting, and the gain in weight has been very satisfactory, a rather high temperature has persisted. This is not commonly found in cases of eczema and one is rather put to it to explain it in this instance. There is no evidence that it is of an acute infectious nature. Certainly, the general condition of the child and his reaction to treatment do not support such a view; nor is there any clinical evidence of such. The only hypothesis which seems to me to be at all tenable is that the temperature is the result of absorption from the skin of the toxic products. This temperature is now showing a tendency to reduce.

There is perhaps no condition more puzzling to the phys-

ician than eczema in children. Many years ago Czerny called attention to the condition, which he has spoken of as exudative diathesis. You will note in this instance that the condition began with a rash on the cheeks and spread from there to the rest of the body. This is quite characteristic of children with exudative diathesis, and in almost every instance in infancy where eczema exists we can make out very definitely the other symptoms of exudative diathesis. But this tells us nothing as to the cause of eczema. It only shows us that a certain class of individuals are prone to this condition. In recent years there has been a tendency to regard eczema as the result of anaphylaxis. Personally I have not been persuaded that this theory is correct. So far as I know eczema has never been shown to be a symptom of anaphylaxis, and the eruptions of a purely anaphylactic nature which do occur are distinctly different from those which we see in eczema. I remember recently seeing a breast-fed baby who had a scaly eczema of the face, but at times this child would break out with a marked eruption which was a generalized erythema in which there would be raised urticarial lesions. This did not materially affect the eczematous condition, and having in view the recent work of Shannon on the presence of foreign proteins in the breast milk of the mother, we thought it wise to treat this condition as an anaphylactic lesion due to something which the mother had eaten. The first attempt made was to eliminate eggs from the mother's diet. The result was most striking. With the elimination of eggs the urticarial lesions entirely disappeared, but there was no effect whatever upon the eczematous process. If anaphylaxis is responsible for eczema one would think that the removal of the protein from the food would be sufficient to produce a cure of the eczema, but, as a matter of fact, the increase of protein elements and the decrease of fats more favorably influences the eczema than does any other means of dietetic treatment. Therefore it does not seem that anaphylaxis could be regarded as being the cause of eczema.

Since our theories of the cause of eczema have not been proved, and since we have no basis at present except an em-



pical one upon which to treat these cases, we must go on the suggestion that anything which will favorably affect the exudative diathesis and reduce the irritation of the skin will materially influence the course. We know from long experience that exudative diathesis is best treated from a dietetic standpoint by the reduction of milk-fat in the food, that these children are capable of taking rather large quantities of starch without producing disturbance, and that the protein content of the food may be relatively very high. Therefore it would seem that from a dietetic standpoint it would be wise to put these children on a protein- and carbohydrate-rich and fat-poor diet.

Now one should not get too exaggerated an opinion of what a change in diet will effect in these children. If we are to rely solely on a change of diet for the results to be obtained we will not obtain results. It is always wise to put these children on such a diet as I have recently indicated. For instance, this baby has one containing 30 ounces of albumin milk,  $\frac{1}{4}$  ounce of dextrimaltose, which has been gradually increased to  $\frac{3}{4}$  ounce, five feedings of 6 ounces each. You will note that he has made a substantial gain and has been without gastro-intestinal disturbance. You will also note (or, perhaps, it would be better to say that I must tell you) that in comparison with the condition in which this child came into us the skin shows such a marked improvement that it is scarcely to be believed. The crusts on the head have disappeared and the harsh, scaly skin has softened, and the redness has gone from every place except the cheeks. The body shows no eruptions and the whole condition is very much improved; in fact, so much improved that one would scarcely believe that such a change could occur in the short space of five days.

Now this change has not been produced by a change of diet, although a change of diet has been made in this instance, but it has been largely produced by certain institutional means of treatment. In the first place, this child was living on a farm. Farmhouses get cold in the night and usually are overheated during the day. The result is that the child is subjected to

changes of temperature which produce marked changes in the superficial circulation of the skin, with the result that any eczematous process is further irritated by the change of blood to these tissues. Next, this child, in a way, was kept too warm. He has been swathed in woolen clothes, and woolen clothes are always irritating to the skin of these eczematous babies. Another probability is that the child was bathed in the ordinary fashion, with soap and water. But, above all, the irritation



Fig. 292.—Photograph showing child on frame.

which the child has produced itself by scratching and rubbing is of far greater importance than all the other modes of irritation. If any of you has had the care of an eczematous baby you will realize how hard it is to overcome this tendency. The children are frantic from the itching. We, therefore, had to change the total environment and care of this baby.

The dietetic change has already been gone into and its effects likewise seen. A change in atmosphere, keeping the child at an even temperature, is, of course, part of the hospital

treatment and one to which we have to pay little attention. However, we should note that it is of special importance, and we may readily realize this when we think that all cases of eczema are far worse in the winter months and have a marked tendency to heal up during the summer months. The child is bathed twice a day with sweet oil. The nurse rubs this in and then applies to the skin an ointment of 2 per cent. ichthyol in Lassar's paste with salicylic acid. This, too, is rubbed into

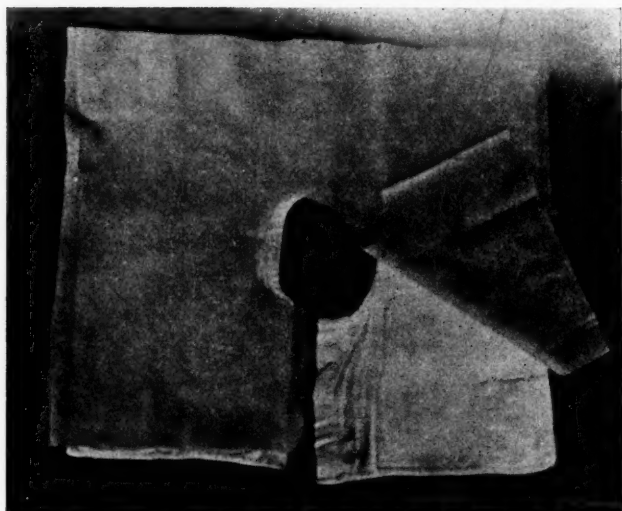


Fig. 293.—Oilcloth bib referred to in text.

the skin, and then the portions of the body except the face are wrapped with sterile gauze. The scalp is treated with sweet oil and as many of the crusts as possible are removed. There is then applied a 10 per cent. sulphur ointment.

Now one might think that these measures are the most important ones from the standpoint of the care of the child. We have already spoken of the diet, the temperature of the room, the method of cleansing the body, and the ointments applied. But of far greater importance than any of these is

the restriction of the child. You will note here that we have a frame to which are attached four screw-eyes, two for the hands and two for the feet. The hands and feet are then loosely tied to these screw-eyes so as to allow the child a certain freedom of motion, but to prevent scratching or turning over. The bedclothes are then tucked in around this frame and about the neck, under the head and under the chin is placed an oil silk bib which effectually prevents the child from rubbing its chin



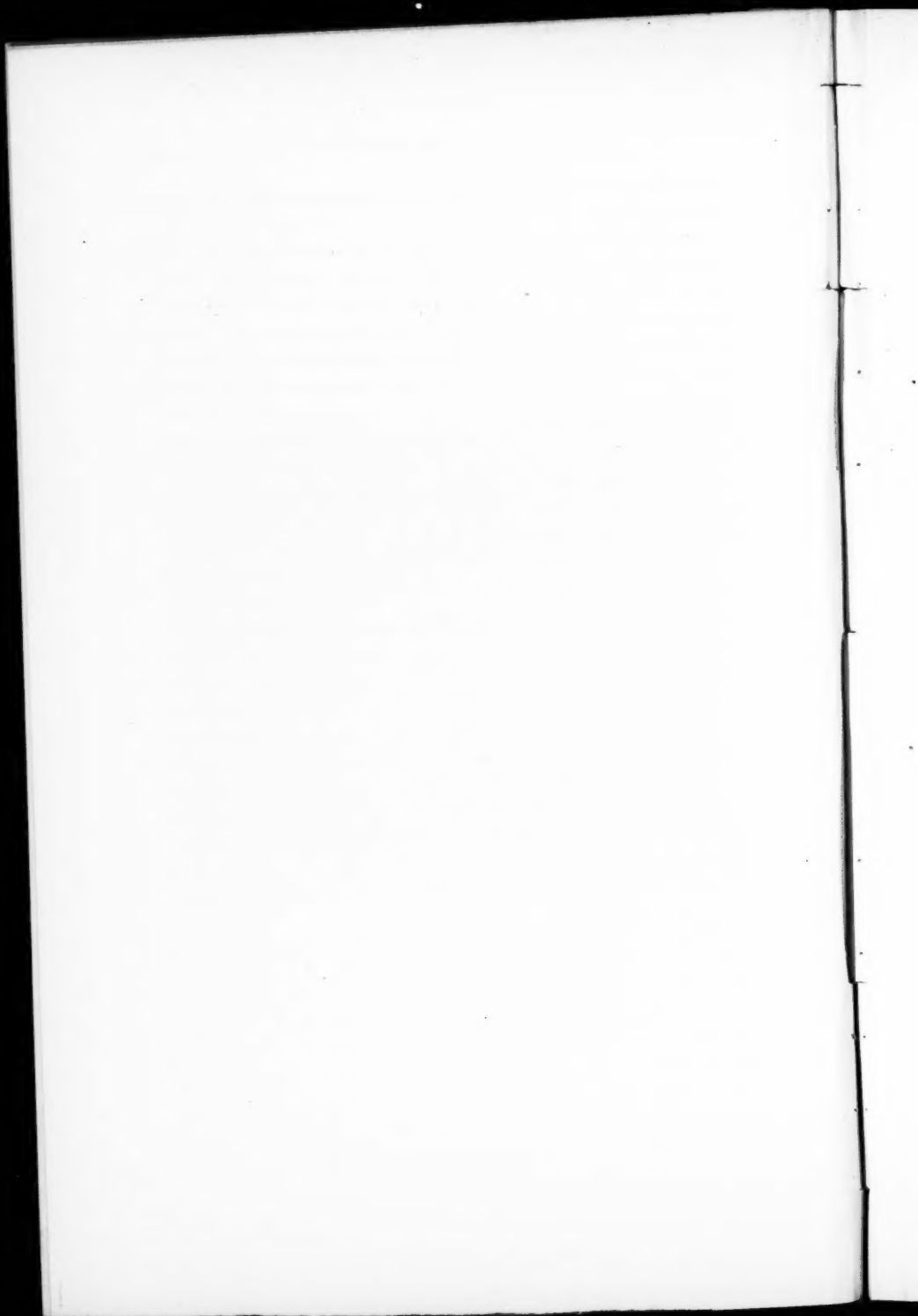
Fig. 294.—Photograph showing child in bed, the frame covered with the bedclothes and the oilcloth bib in place.

or head in such a way as to produce irritation. Coming in contact with the smooth surface of the oil silk there is no friction produced by the skin to which the ointment has been applied, and the result is that no irritation of that skin is produced. Certain people will feel that it is cruel or unwise to place the baby in such a frame. To me it seems that it is the most efficient form of mercy. Within a very short time these children, having been relieved of the irritation of the eczema, become quiet and content to remain thus restricted in their

movements, and certainly the results to be obtained are most gratifying.

I do not wish you to go away from here feeling that this case represents a cure of infantile eczema. It represents nothing of the sort. It only means that we have here a method by which we can control the eczematous process, and by which, if it is applied over a long enough period and with sufficient care, will eventually mean a clearing up of the process and a skin which usually turns out to be very delicate and very fine. We are by no means through with this case. It will take weeks and months before such a result can be expected, but when we realize that this baby has been going along with the most severe eczema for the last two months, it is most gratifying to think that we have been able to get it under control in a few days.

I want to call your attention to the fact that this work has been done in the hospital. If we are to obtain results in these cases in many instances they must have hospital care. It is just as necessary that a severe case of eczema of this type be subjected to hospital care as it is that an appendicitis must come to the hospital for operation. In fact, I believe that it is more necessary. It will require a great deal of education of the public to override the prejudice against hospitals as a place for the care of children, but certainly a properly conducted hospital is of the greatest importance in this respect. It is of great importance to educate the public to see what can be done in hospitals, and I am free to admit that I do not believe it would have been possible to obtain results of this type under any other conditions.



## CLINIC OF DR. ARTHUR F. BYFIELD

COOK COUNTY HOSPITAL

### A. A CONSIDERATION OF DISEASES WITH ASSOCIATED CHANGES IN THE SPLEEN AND LIVER (THE SPLEEN-LIVER SYNDROME).

### B. INDICATIONS FOR SPLENECTOMY IN THIS SYNDROME.

Examples of the Spleen-liver Syndrome; Nature of the Splenic and Hepatic Changes in this Syndrome. Discussion of the Cirrheses of the Liver, Banti's Disease, and Splenic Anemia. Indications for Splenectomy in the Spleen-liver Syndrome.

DURING this period we plan to sort over and rearrange some of the material gathered from previous clinics devoted to conditions in which there have been present associated enlargements of the spleen and liver. This associated pathology we shall speak of as the *spleen-liver syndrome*. Of the conditions belonging to this clinical group, some have apparently exhibited a more or less coincident involvement of the two organs, while others seemed to develop with a primary involvement either of the spleen or of the liver.

As examples of the spleen-liver syndrome we have commented upon, or have studied in detail, many of the acute general infections; the leukemias; the several cirrheses of the liver; splenic anemia and Banti's disease; Hodgkin's disease; hemolytic icterus; anemia pseudoleukemica infantum (v. Jaksch); splenomegaly (type Gaucher), and pernicious anemia.

Excepting only the leukemias and the Gaucher type of splenomegaly we feel it is rational—and from the point of view of therapy desirable—to bring these conditions, despite their diverse



clinical, anatomic, and histologic pictures, into a closer relationship than has heretofore been the custom. The common basis, if such there be, is, we believe, their pathogenesis, or mode of production.

Gaucher's splenomegaly is probably an anomaly (tumor?) of the reticulo-endothelial apparatus, while the leukemias do not lend themselves readily to classification. Aside from these two conditions, however, it appears reasonable to catalog the several diseases mentioned either as (a) acute infections, (b) chronic infections, or (c) toxemias, chiefly chronic in character.

Our aim in this clinical period is to review first the nature of the associated hepatosplenic changes in the conditions we have placed in the syndrome under consideration; second, to enter at some length into the pathology—particularly clinical—of the liver and spleen in the several cirrhoses of the liver and in splenic anemia and Banti's disease; and lastly, to attempt to construct a rational basis for splenectomy in these splenomegalic conditions.

**A. The Nature of the Splenic and Hepatic Changes in the Spleen-liver Syndrome.**—1. *The Acute General Infections.*—In a great many of the acute general infections there is a variable degree of enlargement—usually moderate—of the liver and spleen. So far as one can determine clinically the enlargement of the two organs is coincident. The nature of the enlargement is one of cloudy swelling due either to the organism producing the infections or to its toxins, or to both.

Typhoid fever, sepsis in its several forms, the acute exanthemata, perhaps malaria in its more acute stages, and many cases of pneumonia, influenza, and meningitis will serve as examples of this type.

2. *The Leukemias.*—Here we encounter a more difficult field, because we are still quite ignorant of the nature of these diseases. It is possible that the acute forms are infectious in character, for there are unquestionably many points of contact between them and known acute infections; and yet, granting this fact, the splenic and hepatic changes in the acute leukemias are, principally, not those just spoken of in connection with the acute infections, namely, cloudy swelling, but are the result of

actual infiltration of the splenic and hepatic tissues by cells characteristic of the blood of the acute leukemias.

The same is true of the chronic leukemias, myeloid and lymphoid. In these the splenomegaly and the hepatomegaly are the result of an invasion of these organs by cells similar to those which enable us to diagnose these diseases from the blood-smear. Sections in the one type show lymphocytic infiltration; and in the other infiltration by cells of bone-marrow origin—myeloblast, myelocyte, and polymorphonuclear.

3. *Hodgkin's Disease*.—Though the pathogenesis of this condition is still unsettled, the bulk of the evidence supports the hypothesis that it is an infection. The granulomatous tissue which characterizes the disease is, as you know, specific; in other words, no other condition produces a similar microscopic picture. It is the infiltration of liver and spleen by this specific granulomatous process—endothelial proliferation, Dorothy Reed type of giant-cell, eosinophil increase, fibrosis, etc.—which causes the enlargement of these organs, the splenomegaly generally antedating the hepatic involvement.

4. *Pernicious anemia* is, as we have said in previous clinics, probably a chronic toxemia due to an agent of hemolytic character which affects primarily the bone-marrow. It is not unlikely that the toxin is not the same in all cases; we know, for example, that the toxins of syphilis and of certain intestinal parasites may produce clinical pictures indistinguishable from that of the so-called idiopathic cases, and it is possible that bacterial toxins of several organisms may do the same. Perhaps, in addition to a given toxin, a certain congenital weakness of the bone-marrow tissues may be a factor.

A clinical enlargement of the spleen and liver can be demonstrated in about one-half of the cases of pernicious anemia; anatomically, a considerable majority of all cases show tumefaction of these organs.

In this disease it is probable that factors quite different from the various ones adduced in explanation of the splenic and hepatic tumors occurring in the conditions already discussed are at work. In the first place, the enlargements are in part toxic

reactions. Second, and of more specific character, the augmented red blood-corpuscle destruction, which is an integral part of the disease and which occurs particularly in the spleen, accounts to a very definite degree for splenic enlargement; while the resulting liberation of iron pigment, giving rise to the so-called hemosiderosis of the organs, contributes to some extent to the enlargement of spleen and liver. Finally, and in our opinion, an important and fundamental factor in the enlargements rests upon embryologic grounds. In intra-uterine life the liver primarily, and the spleen to a less degree, are the most important sites of blood formation. Toward the end of fetal life or perhaps soon after birth this function is shifted to the bone-marrow. Now, when, as in pernicious anemia, the bone-marrow becomes exhausted, the reserves—*i. e.*, the liver and spleen—are pressed into service, and these organs enlarge in response to the unusual work thrust upon them.

Inasmuch as increased red blood-cell destruction is an integral part of pernicious anemia, and as this destruction occurs chiefly in the spleen, we have reason to assume that splenic enlargement is not principally due to this cause, because the splenomegaly is not constant and because the enlargement varies so greatly in the different cases. It seems more rational to believe that the enlargement both of the liver and spleen depends more particularly upon the toxic factors at work and upon the ability of these organs to resume their embryologic function of red cell formation. The bearing of these points will appear in our discussion of splenectomy in pernicious anemia.

5. *Hemolytic icterus* is also, in all likelihood, a chronic toxemia, and as such falls easily into our classification based upon pathogenesis. We have already discussed the clinical features of the two recognized types of the disease—the congenital and the acquired—and have pointed out the occasional rather close similarity of the blood-picture of the acquired form to that of pernicious anemia.

In view of the fact that the acquired type does not exhibit the same lowered resistance of the red blood-cells to hypotonic salt solutions as does the congenital, it is probable that the sple-

nomegaly common to both forms is not due in the main to increased red cell destruction, but rather to the action of the hemolytic toxin supposedly responsible for the process.

The splenic enlargement in hemolytic icterus is generally quite disproportionately great in comparison with the increase in size of the liver; the latter, in fact, is often not enlarged at all. This fact, as we shall see later, is of importance in making hemolytic icterus one of the diseases especially benefited by splenectomy.

At this point it is worth while to note the contacts formed by hemolytic icterus, on the one side with pernicious anemia, *i. e.*, in the matter of the severe anemia of some of the acquired forms, often of a type greatly resembling the blood of pernicious anemia and in the matter of a toxin of hemolytic nature thought to be common to both; and, on the other side, with the cirrhoses of the liver, splenic anemia, and Banti's disease, in which, as we shall point out later, it would also seem that a toxic agent of varying virulence and variable action is responsible. The view, once widely held, that hemolytic jaundice is really pernicious anemia—on account of certain similarities in the blood-picture—is no longer tenable; but the view that hemolytic icterus is a constituent of the large group of the hepatic cirrhoses, as we shall presently define this, is not at all improbable.

6. The etiology of the *v. Jaksch type of infantile anemia* is quite unknown. The blood-picture suggests a stormy reaction of the bone-marrow, both leukopoietic and erythropoietic. Inasmuch as the disease is generally associated with or follows an infectious or toxic process, it is fair to assume that infection or toxemia, or both, play an important rôle in producing the condition. By some observers *v. Jaksch's anemia* is regarded as the infantile form of splenic anemia, the unusual blood-picture being attributed to the unstable bone-marrow equilibrium characteristic of early life.

7. *Splenomegaly of the Gaucher type* is probably either an anomalous constitution of the endothelial apparatus or an actual new growth of endothelial character. It is of congenital and familial occurrence and is associated with a marked enlargement of the liver. Nests of the characteristic endothelial cells

may also be found in other organs—lymph-nodes, bone-marrow, etc. In other words, the process shows a tendency toward generalization.

**B. The Cirrheses of the Liver, Banti's Disease and Splenic Anemia.**—As we stated at the outset we shall discuss these under one of the main divisions of our theme rather than as a subhead of the preceding division. We are thus emphasizing the hepatic cirrheses, splenic anemia and Banti's disease, because we feel that a thorough understanding of these diseases best orients one in the matter of the liver-spleen syndrome, especially from the point of view of pathogenesis and treatment.

The term "cirrhosis of the liver" brings to mind immediately the portal or Laennec form and the biliary or Hanot type. The usual pathologic and clinical pictures of these two forms are so well known to all of you that we may dismiss them with reference to a very few points in the differential diagnosis. We shall consider the more important difference in the two diseases under the following headings:

1. *Alcohol.*—In the Laennec form a history of overindulgence in alcohol can usually be elicited; in the Hanot form rarely.

2. *Age.*—The Laennec type is essentially a disease of middle life, the Hanot, more often of early adult life.

3. *Liver.*—The typical liver of alcoholic cirrhosis—the hob-nailed liver—is atrophic, hard, finely nodular, with a sharp margin. Often in the early stages of the disease and in cases in which well-marked fatty changes go hand in hand with the cirrhotic, the liver is enlarged, but nevertheless firm and with a sharp margin. In biliary cirrhosis the liver is consistently large, firm, and with a margin likely to be only moderately sharp.

4. *Spleen.*—The spleen of alcoholic cirrhosis is always enlarged, even though clinically in a certain number of cases it cannot be palpated. The enlargement is typically moderate. In the Hanot type, on the contrary, a large, firm, smooth spleen is characteristic.

5. *Jaundice,* if present at all in the alcoholic form, is slight and usually late. Often it is of the form called subicteric, the urine generally giving no test for bile. Marked icterus in the

alcoholic form is usually due to a complicating cholangitis. In the Hanot type, on the contrary, there occur periods of intense jaundice, associated often with fever and leukocytosis.

6. *Ascites* in the alcoholic form is fairly early; in the Hanot form, terminal.

These are some of the more striking distinctions between the usual types of these two diseases. However—and this is a very important point in our argument—there are observed not infrequently mixed types of these two cirrheses, anatomically verified, but clinically usually only suspected. Thus there may be an unusually large spleen in a case otherwise conforming to the Laennec type; or there may be an unusually large liver, with or without an unusually large spleen in a case apparently belonging to the alcoholic group; or still other mixing of the types. Anatomically such cases show usually hepatic changes in part characteristic of the one form, in part of the other.

To sum up at this point, therefore, we may say that the generally accepted hard-and-fast distinctions between these two types of cirrhosis are more apparent than real, and that there is an underlying factor—infections or toxic, or both—common to the two which produces clinical and anatomic pictures that would appear to be widely different.

If we are granted the validity of this argument we may pass without a break in our theme to a consideration of splenic anemia and Banti's disease. In the light of our present-day pathologic conceptions these two conditions must be considered as one, splenic anemia being only an early stage of Banti's disease, in which cirrhotic changes in the liver, and later, ascites, are observed.

Now let us see just what we mean when we speak of splenic anemia and its later stage, Banti's disease. Splenic anemia, clinically, is a disease beginning, as a rule, in young adult life, more commonly in males, and characterized by a large spleen, anemia of the secondary type—associated with a leukopenia and often a relative lymphocytosis—and with a tendency to hemorrhages from mucous membranes, perhaps oftenest from the mouth. Pathologically the chief characteristics are an endo-

phlebitis of the splenic vein extending in variable degree toward the larger portal vessels, plus fibrous changes in the spleen and its capsule. As the disease progresses the second stage supervenes—that of Banti's disease—with hepatic cirrhosis and terminal ascites.

If we add to what we have just said that the process comes on without apparent cause, and, in particular, is not due to alcohol, we have the substance of Banti's own description of the disease.

Now, as often happens in the case of a supposedly well demarcated pathologic condition, further experience shows that the disease as originally described has been too strictly walled off. It does not always run true to type; it overlaps other diseases at one point or another. This is true of Banti's disease and the cirrhoses of the liver. Note the following points of contact:

1. Cases which clinically have all the accepted earmarks of splenic anemia and Banti's disease and which pathologically fulfil Banti's postulates have been proved to be due to alcohol, syphilis, malaria, tuberculosis, and other infections. It has been suggested, therefore, that we speak of Banti's symptom-complex rather than of Banti's disease.

2. Cases which are clinically Banti's disease prove at autopsy to have anatomic findings characteristic of one of the usual types of cirrhosis, and in particular not the specific splenic changes of Banti's disease previously mentioned. You will recall that we have already made note of the fact that certain cases of what appear in other respects to be typical Laennec cirrhosis exhibit unusually large spleens.

3. The terminal stages of all are practically identical.

Though there are still other arguments along this line that might be adduced, we believe these three are sufficient to establish our point, namely, that the cirrhoses of the liver and splenic anemia and Banti's disease approach one another closely at many places. Indeed, we have gone so far as to say that they are, to all intents and purposes, one large pathologic process with variable manifestations.



Relative to this point we might express our views as follows: The cirrheses of the liver, splenic anemia, and Banti's disease are all due to a toxic or infectious process, or to a combination of the two. The type of the infection or toxemia, its predilection for certain organs, and its effect upon these organs vary with the case.

In alcoholic cirrhosis the toxic agent—incidentally, as you well know, this is probably not the alcohol itself, but some other poison the formation of which the alcohol favors—acts primarily upon the liver and to a much less degree upon the spleen (syphilis, tuberculosis, malaria, and other infections, in the absence of alcohol, may produce clinical and pathologic pictures indistinguishable from the alcoholic, another argument emphasizing the blending of the several members of the cirrhosis group). Nevertheless, as we have seen, the toxemia may affect the spleen to an extent almost as great as it does the liver; these are the cases of Laennec cirrhosis with unusually large spleens. In the biliary or Hanot form the infectious or toxic-infectious agent, as the case may be, affects the liver and spleen in practically equal degree. In what we have called the mixed cases of the two types, spleen and liver exhibit transitional effects of the causative factors. Inasmuch as the actual causative factor, or factors, are not known, it is scarcely worth speculating as to just how the mixed pathology is produced.

In splenic anemia and Banti's disease, on the other hand, the toxic agent shows a primary predilection for the spleen, the liver being secondarily affected. In our opinion, therefore, conditions stand somewhat as follows: Alcoholic cirrhosis of the liver and Banti's disease are both manifestations of a toxemia of unknown origin; in the one the toxic agent attacks first the liver, in the other the spleen. Biliary cirrhosis apparently occupies a position midway between these two.

Returning now to the question of hemolytic icterus we find it justifiable if our theorizing is correct to include this condition in our larger group of the cirrheses. As a matter of fact, cirrhosis is not a good name for the several conditions we are considering; it is a traditional name and there is nothing more

difficult in medicine than to discard tradition. A more correct term would be the "toxic-infectious liver-spleen syndrome." Hemolytic icterus, as you will readily grant, belongs in the latter collective grouping, for it is nothing more than a toxemia in which a poison of hemolytic character affects primarily the spleen.

At this point we may crystallize our theme as follows: First, there are a number of diseases with separate names which really are only members of one large group—the toxic-infectious liver-spleen syndrome; second, the indications for splenectomy rest upon well-established grounds if this hepatosplenic syndrome is well understood.

We shall discuss the second main point—the indications for splenectomy—first, and this we can best do by considering once more the several conditions with enlarged spleen and liver, *i. e.*, those mentioned at the beginning of our remarks, in the light of what we have said concerning the cirrhosis group of diseases.

**C. Splenectomy is indicated** in those cases of chronic infection, chronic toxemia, or in combinations of infection and toxemia, in which the primary and predominant action of the etiologic factor is upon the spleen. Put in other words, splenectomy is indicated in those cases in which the spleen seems to become the main distributing point of the toxic or infective agent.

Viewing the liver-spleen syndrome in this light it is not difficult to pick out from our original list of conditions belonging to this syndrome those in which removal of the spleen may, with some hope of success, be advised. Parenthetically, we may add that it should now appear why, earlier in our remarks, we went into detail as to pathology and clinic in discussing the diseases we are about to consider from the standpoint of splenectomy.

1. *The Acute Infections.*—It is almost absurd, of course, even to mention the acute infections in a discussion of splenectomy. The splenic enlargement in these conditions is, first of all, transitory—as is the disease itself; and in the second place it is only a subordinate feature of the disease process as a whole. We may, therefore, dismiss these conditions without further discussion.

2. *The Leukemias*.—As we have already noted, the acute leukemias bear a close resemblance in many ways to the acute infections, and for reasons stated in connection with the latter do not offer a suitable field for splenectomy. But there are more important contraindications to the operation, these contraindications applying with equal force to the chronic leukemias.

The *leukemias*—and what we have to say applies especially to the myeloid form—even in those cases in which the spleen is unusually large (sometimes called the splenic form of myelogenous leukemia) are not diseases of the spleen. They are not conditions in which the etiologic factor, whatever that may be, makes its home in the spleen. Leukemia is a disease of the bone-marrow or of the lymphatic tissues, as the case may be; and the enlargement of the spleen means only that the bone-marrow, or lymphoid elements, of the organ have proliferated as they have elsewhere in the body; added to which, in all probability, there is an invasion of the spleen, by the same cells, from the blood-stream. It would be just as logical to remove the liver. The mere fact that in a particular case the spleen is unusually large has no special significance.

You will recall that splenectomy was advised by certain writers in the recent past, upon what scientific grounds we do not know. It was always our belief that no good could come from the operation in these cases, and we believe that experience has justified our belief. So far as we know splenectomy is no longer to any extent being advised in the leukemias.

What has been said of the leukemias applies in large part to *pernicious anemia* insofar as the bone-marrow seems to be the organ of chief involvement. In pernicious anemia, also, even at the height of the enthusiasm for splenectomy, it was our opinion—and we so went on record in 1918—that no hope of permanent success need be awaited from the operation. This opinion we based upon the well-established ground that the essential pathology of the disease resided in the bone-marrow. As we have seen, the splenomegaly is probably due to several factors. If a large part is due to compensatory erythropoietic

activity, removal of the organ is surely unwise. If the essence of the disease is an increased red-cell destruction, removal of the spleen might be a step well taken, but as the augmented destruction is only one factor in the disease, splenectomy can only work a temporary benefit in the way of allowing the bone-marrow to "catch up" as it were. And this is what actually seems to take place in those cases which have apparently responded favorably to the operation. However, transfusion accomplishes the same end and is, for evident reasons, the operation of choice.

In *Gaucher's disease* splenectomy has been performed a number of times, in several instances apparently with benefit. However, the generalization of the process, its nature, and its chronicity would seem to offer little real hope of even fairly permanent success.

As for the *v. Jaksch type of anemia*, splenectomy seems rational in certain instances. Many cases, as you have been told, go on to a good recovery under medical measures alone. In those patients, however, who do not improve after a fair medical régime, in which the anemia progresses and the splenic enlargement increases, removal of the spleen is definitely to be considered. These are the cases which touch shoulders with the group in which splenectomy is clearly advisable—the toxic-infectious hepatosplenic enlargements.

So far as we know splenectomy has not been considered in *Hodgkin's disease*. In the first place, the latter is always a general process; and in the second, even though a splenomegaly may dominate the picture there is always an associated—and usually primary—involvement of lymph-nodes, generally cervical. As well, therefore, to operate upon the metastases of a carcinoma as to remove the spleen in Hodgkin's disease.

We come finally to the toxic-infectious liver-spleen syndrome in which splenectomy has found its greatest success. Even here, however, the indication is present only in the selected case. Splenectomy in this group is indicated under the following conditions:

1. In cases in which, under the influence of a chronic infection

or chronic toxemia, the spleen is primarily and predominantly involved, acting, as it were, as the main depot of the infection or toxemia. Splenic anemia in its earliest stages and hemolytic icterus are the conditions par excellence in which the operation is successful.

2. In the Banti group of diseases splenectomy is particularly indicated if the etiology is obscure, but even in cases in which the cause can be determined—syphilis, tuberculosis, malaria, focal disease elsewhere—if proper specific therapy proves unavailing, the operation is still indicated if the spleen stands out as the central point of the toxic-infectious picture.

3. Slight enlargement of the liver, with indications 1 and 2 present, and the contraindications about to be mentioned absent, is not in itself an absolute contraindication. In such cases removal of the main depot of mischief—the spleen—may produce a recession of the liver changes, or at the least may prevent further hepatic damage.

4. In the absence of certain general conditions which contraindicate operation under any circumstances. These are the cardiac, renal, pulmonary, and other diseases in the presence of which operation is to be avoided unless there is a life-threatening emergency.

We believe that the above-mentioned indications for splenectomy in what we have called the liver-spleen syndrome are conservatively drawn, and based upon good pathologic grounds, and our experience with this type of cases has been such as to justify the soundness of our position.

By way of résumé, then, we may say:

1. The spleen-liver syndrome, so-called, embraces a number of pathologic processes, largely unrelated, but in the main due to toxic or infectious causes.

2. The term "cirrhosis of the liver" appears to be too narrow in its application, as there seem to be good reasons to ally with the cirrhoses, in the narrow sense, splenic anemia and Banti's symptom-complex and hemolytic icterus,

3. A study of the pathogenesis of the various members of the spleen-liver syndrome, plus clinical experience, seems to

show that splenectomy is indicated only in the cirrhosis group of diseases—using this term in its larger sense as we have defined it—and then only in the presence of certain definite indications and in the absence of certain contraindications.

## CLINIC OF DR. JAMES G. CARR

COOK COUNTY HOSPITAL

### BANTI'S DISEASE

**Patient Presenting Symptoms of Banti's Disease. Differential Diagnosis Between Atrophic Cirrhosis, Banti's Disease, Syphilitic Cirrhosis, and Hanot's Disease. Treatment: Splenectomy, with Good Recovery. Pathologic Report of Specimen Removed.**

THIS young man, to whom I wish to direct your attention this morning, was admitted to our service September 27, 1921. He is twenty-three years of age, a native of Poland, and by occupation a laborer. He is married; his wife and one child are living and well; one child died at seven months; the cause of this death cannot be ascertained. His wife has had no miscarriages.

The patient came to the hospital with the following principal complaints: Pain beneath both costal margins and across the lower part of the abdomen; constipation; blood in the stools; a tumor beneath the left costal margin; a history of hematemesis and of undue bleeding when the skin is injured.

He has not been well for eighteen months or more, stating that his present illness dates from an attack of influenza at the time of our last epidemic in the early months of 1920. For several months thereafter he suffered with pain in the right lower quadrant which was relieved by movement of the bowels. This pain was practically constant; at times there were acute exacerbations, about which we can get no accurate reports from the patient.

On February 15, 1921 his appendix was removed. Since the operation there has been no improvement; rather he has been incapacitated to an even greater extent than before. There has

been marked constipation necessitating the frequent use of cathartics. He has noticed a dull dragging pain under the left costal arch; this pain is often worse after meals; it is aggravated by hard work or bending forward. Occasionally there is a similar pain on the right side. These pains are often accompanied by nausea. There have been infrequent spells of vomiting. Five months ago he vomited a large quantity of blood; there were several attacks of hematemesis over a period of two or three days. The immediate cause of his coming to the hospital was a second gastric hemorrhage; for two or three days prior to his admission he vomited blood; in one attack, so the patient says, he almost filled a wash-basin with blood. The hemorrhages from the bowel, mentioned in the first summary of his important symptoms, appear not to have been of intestinal origin, but confined to the passage of blood per rectum following the gastric hemorrhage. The patient has a taste for food, but fears to eat because of the ensuing distress. His strength is not equal to the necessities of his work, though he has not kept to his bed. He does not know whether or not he has lost weight.

His past history is negative; the family history unimportant. Venereal disease is denied. Tobacco has been used in moderation; alcohol not at all.

Physical examination shows a moderately well developed, rather poorly nourished man of a slightly anemic appearance, with his skin showing a faintly icteric tinge. (This statement is taken from the original record made on admission. The attending physician did not concur in the opinion that icterus was present.) The temperature at the time of admission was 98.3° F., pulse 76, respirations 20, blood-pressure 120/65. There was no general adenopathy. The reflexes were normal except for equal exaggeration of the patellars. No abnormalities of the blood-vessels were noted. The teeth were in generally good condition; there was one decayed tooth which needed attention. Two small hemorrhoids were found. The genitalia were negative.

Both the right and left lobes of the liver were easily palpable; the right lobe could be felt about two fingerbreadths below the costal arch in the mammary line. The edge was sharp



and firm, the surface smooth; there was no tenderness on palpation. The spleen was definitely enlarged, firm, and smooth; with quiet breathing it could be felt full three fingerbreadths below the costal arch. There was no tenderness. There was no evidence of ascites. Neither kidney could be felt. No abnormalities were noted in the cardiac examination. The lower borders of the lungs appeared unusually high on both sides, posteriorly and in the axillary regions. These latter findings were attributed to the hepatic and splenic enlargements. Except for a few moist râles about the hilus of the right lung and over the area about the lower angle of the right scapula pulmonary examination was negative.

The provisional diagnosis as recorded by Dr. Leffert called for the differentiation of (1) atrophic cirrhosis, (2) Banti's disease, (3) syphilitic cirrhosis, (4) Hanot's disease. The last mentioned condition has not been seriously considered in the further development of our diagnosis because of the absence of unquestioned icterus. Hanot's disease, a rare condition, characteristically presents distinct jaundice. For the same reason hemolytic icterus was not seriously considered in the differential diagnosis. On October 6th the fragility test was made; hemolysis did not occur in solutions of greater strength than 0.36 per cent. of NaCl. This was accepted as finally excluding hemolytic icterus.

Before we undertake any further discussion of the differential diagnosis it may be well to review the course of the disease, since the patient has been under our observation and to cite to you the results of our laboratory and roentgenologic examinations.

Since the patient's admission to the hospital only twice has the temperature been recorded as above 99° F.; there is one reading of 99.4° F. and another of 99.2° F. The pulse-rate has varied from 70 to 90; the respirations have been about 20 per minute. The urine is negative.

Upon admission the blood count showed 4,500,000 reds, 2500 whites, and a hemoglobin percentage of 92. The differential leukocyte count showed small mononuclears 26, large mononuclears 8, polymorphonuclears 66. No nucleated red cells

were found. On October 5th the reds were 4,760,000, whites 2700, small mononuclears 20, large mononuclears 2, polymorphonuclears 78. On the 10th the total red count was 4,800,000, total white count 2600, with differential figures thus: small mononuclears 28, large mononuclears 12, polymorphonuclears 60. On October 24th the hemoglobin was 56 per cent., the red count 4,370,000, the white count 2100. Differential count showed polymorphonuclears 60, small mononuclears 30, large mononuclears 6, eosinophils 2, basophils 2.

The blood Wassermann was negative. An Ewald test-meal was given October 5th; the free HCl was 28, total acidity 56. Two days later the test was repeated; about 200 c.c. of gastric contents, largely blood, were removed. No further attempts to examine the gastric contents were made.

On October 4th the roentgenologist reported absence of any evidences of pathology of the gastro-intestinal tract. On October 19th chemical examination of the blood showed per 100 c.c. of blood: chlorids 675 mgm., non-protein nitrogen 40.21 mgm., urea nitrogen 21 mgm., urea 46.97 mgm., uric acid 1.85 mgm., creatinin 1.51 mgm., sugar 134 mgm. Carbon dioxid tension of the blood was 61.4 per cent.

Here it might be well to collect the important facts from the history and physical examination, so that we may have clearly before our minds the significant features of the case. We present to you a young man who has not been well for some twenty months, who has lost strength without much loss of weight, who has suffered vague abdominal pain, mostly localized in the left hypochondrium. There have been two large gastric hemorrhages. There is enlargement of the liver and of the spleen, with a moderate secondary anemia, with leukopenia.

From the standpoint of diagnosis the outstanding features of the case are the gastric hemorrhages and the splenic enlargement. In accounting for these, alone or together, it is probable the road to diagnosis lies.

What was the source of the gastric hemorrhage? Ulcer or malignancy must be considered. Aside from the history of hemorrhage we have evidence neither for ulcer nor cancer. The

symptomatology is not suggestive of ulcer, the usual points of tenderness so common in ulcer are not found, the single successful gastric analysis showed no deviation from normal secretion; finally, the roentgenologic examination showed no "evidence of pathology." Like statements may be made regarding the presence of cancer; neither the history, physical findings, laboratory nor x-ray findings lend support to such a diagnosis. Furthermore, we feel justified in expressing the view that, in the absence of distinct and clear-cut findings the diagnosis of carcinoma in a patient of twenty-two is unwarranted, and sarcoma is a pathologic curiosity rather than a clinical probability. In spite of the patient's statement when his history was first written that he had shown a tendency to bleed excessively from small skin wounds, we have not been able to elicit further evidence of any one of the so-called hemorrhagic diseases.

The other frequent cause of hematemesis is enlargement of the esophageal veins. These varices occur with portal obstruction; they are especially characteristic of atrophic cirrhosis and of the symptom-complex known as Banti's disease. In connection with the latter, and with the particular type of splenomegaly known by the name of Gaucher, it seems probable that gastric hemorrhages sometimes antedate the development of varices; it is assumed that these hemorrhages are toxic in origin.

What is the significance of the enlarged spleen? The diagnostic possibilities may be said, in this case, to be confined to syphilis, malaria, atrophic cirrhosis, or Banti's disease. Some forty years ago Banti described as a clinical entity a symptom-complex which has since gone by his name. This symptom-complex included splenomegaly, secondary anemia without leukocytosis; after a period of variable duration there occurs hepatic cirrhosis with ascites, often associated with gastric hemorrhage.

From the time of his original description Banti's disease has been subjected to much discussion and criticism. By many "splenic anemia" has been accepted as synonymous with Banti's disease; Banti himself recognized the similarity of the two conditions, but regarded them as distinct. In text-books they are usually discussed under the same heading. In my student days

this division into three stages was given to us: (1) splenomegaly, (2) splenic anemia, splenic enlargement with secondary anemia without leukocytosis, the second stage, (3) splenic anemia plus cirrhosis of the liver and ascites, the third stage or Banti's disease. We may add that splenic anemia or Banti's disease is only entitled to consideration as a clinical entity, when the symptom-complex occurs without demonstrable cause.

Syphilis may produce symptoms identical with those presented by this patient. The symptom-complex of atrophic cirrhosis or of Banti's disease may be exactly simulated by syphilitic disease of the liver and spleen. A perfect picture of Banti's disease may occur with definite evidence of its syphilitic origin. We shall discuss in more detail later the justification for the use of the term "Banti's disease"; much of the discussion of this term has been excited by two facts: (1) the identity of certain types of splenic and hepatic syphilis with the clinical picture of Banti's disease; (2) the resemblance of Banti's disease to atrophic cirrhosis. There is something to be allowed to those who assume that, after all is said, Banti's disease may be regarded as "atrophic cirrhosis backward." It is altogether possible that the same underlying cause, toxic or infectious, may produce predominant symptoms of atrophic cirrhosis or of splenic anemia; in each case both liver and spleen are involved, presenting a clinical picture, finally much the same, whether the early symptoms were mainly hepatic or mainly splenic.

In the case before us syphilis may be excluded; there is no history of syphilis; clinical evidence is lacking; the Wassermann is negative. There is no proof to support the diagnosis of specific disease. Neither is there any evidence that the patient now has or has ever had malaria.

Are we dealing with a case of Banti's disease or of atrophic cirrhosis? Favoring the diagnosis of atrophic cirrhosis are these findings: hepatic enlargement, splenic enlargement, gastric hemorrhages. Hepatic enlargement is mentioned here rather out of deference to accepted opinion than from any personal conviction. I am not convinced that enlargement of the liver, demonstrable on palpation, is an early clinical sign of atrophic

cirrhosis. In this connection a statement of Leube's is recalled; in one of the editions of his work on diagnosis this author says the statement is customarily made that the liver is enlarged in the early stages of atrophic cirrhosis; no such enlargement had ever been observed by him.

Against the diagnosis of atrophic cirrhosis we have: (1) the age of the patient, (2) absence of the characteristic "dyspepsia," (3) early and disproportionate enlargement of the spleen, (4) the progressive anemia of secondary type, with an accompanying leukopenia. There has been a steady and pronounced fall in the hemoglobin; the most recent red cell count, made since the patient's transfer to the surgical ward, showed 4,360,000 cells with 2100 leukocytes and a hemoglobin percentage of 56; the highest white count recorded at any time was 3500. The preponderance of evidence is against the diagnosis of atrophic cirrhosis; the same findings, on the other hand, are those which are commonly regarded as characteristic of Banti's disease, whatever that may be.

No reference has been made to the absence of demonstrable ascites. The absence of ascites is of no significance either way. The presence of ascites with hepatic cirrhosis is regarded as characteristic of the third stage of Banti's. If ascites were here present it might be adduced as further evidence against atrophic cirrhosis. Whether or not the liver shows enlargement early in atrophic cirrhosis, it is certainly true that ascites, in atrophic cirrhosis, is usually found only after the cirrhotic process is fairly advanced and the organ decreased in size. Perhaps the absence of ascites along with the marked enlargement of the spleen speaks for splenic anemia as against atrophic cirrhosis.

Reviewing our consideration of the differential diagnosis, we come to the conclusion that of the diagnostic possibilities mentioned, Banti's disease is the one which covers the findings as stated. The findings in this case may be regarded as classically those of splenic anemia or Banti's disease. Save for the moderate grade of anemia they are classical. Gradual loss of strength, splenomegaly, enlargement of the liver, gastric hemorrhages, secondary anemia with leukopenia, all of these findings

without a discoverable cause make up a picture only to be described as Banti's disease.

Of recent years the literature has been rich in discussions of Banti's disease, its nature, pathology, pathogenesis, symptoms, diagnosis, and therapy. It is at present altogether unwise to dogmatize about "Banti's disease"; its place as an established clinical entity is not secure. Two assertions may be made: (1) Banti's original description included one group which presents characteristic features and can be classified as a separate entity; this is known as the Gaucher type; (2) the clinical complex and the pathology described as characteristic of Banti's disease may occur as the result of syphilis, malaria, thrombo-phlebitis of the splenic or portal veins, or of other conditions.

The Gaucher type is one which may be diagnosed clinically with a very considerable degree of certainty. Anatomically it presents characteristic large cells, endothelial in character, not confined to the spleen, but found also in the liver, lymph-glands, and bone-marrow. The disease is of long duration, often beginning in childhood; the spleen may be greatly enlarged; there is a tendency to subcutaneous hemorrhage and hemorrhage from the gastro-intestinal tract; a secondary anemia without leukocytosis is present. Clinically and anatomically the Gaucher type can be distinguished from the heterogeneous "splenic anemias" included under the term "Banti's disease."

The latter group still is heterogeneous. Malaria and syphilis, especially the latter, produce symptom-complexes in no way different, clinically or anatomically, from the disease described by Banti. Five years ago Giffin, from the Mayo Clinic, described in detail 3 cases of splenomegaly with secondary anemia without leukocytosis. Two of these cases showed while under his observation positive Wassermann reactions. The third had shown on three occasions previously a positive reaction, but the test was negative while the patient was under Giffin's observation. Splenectomy was done in these 3 cases, with prompt improvement in all. Microscopically these spleens all showed diffuse fibrosis; in one typical gummata were found; in the

others a few small gummata and treponemata. Norris, Symmers, and Shapiro have gone so far as to make the following statement: "It will be shown, we think, that Banti's disease as an entity has no legitimate claim to recognition, and that the designation is an encumbrance in the nomenclature of splenic diseases; that it really represents a splenic manifestation of syphilis usually with, but occasionally without, concomitant sclerotic changes in the liver, and that all the clinical and anatomic requirements of the disease as originally postulated by Banti are adequately satisfied by syphilis."

I am not willing to concur in the opinion just quoted. In the contribution from Giffin just mentioned a fourth case was reported in which there was a history of syphilis ten years prior to the time the patient came under Giffin's observation; Giffin found a splenic anemia, a negative Wassermann, and no evidence of syphilis on section of the spleen. In the case before you we have the clinical picture of splenomegaly, secondary anemia without leukocytosis, and hematemesis, without any evidence of syphilis. The time may come when the designation "Banti's disease" will be discarded; I incline to the belief that when such a time does come the generic term will be dropped because the splenic fibrosis will be capable of fairly exact classification on the basis of etiology; we will classify splenic fibrosis or cirrhosis much as we now classify the various types of cirrhosis of the liver.

Other causes than syphilis may produce the pathologic changes in the spleen regarded by Banti as characteristic of the disease he described. Malaria has been mentioned. Dock and Warthin described a good many years ago the occurrence of thrombosis of the portal vein in connection with splenic anemia. Naegeli regards the splenic change as a secondary process, stating that the sclerosis is in itself a non-specific change. He says further, "The majority of the most experienced hematologists agree that they have never seen a case in which clinically and anatomically 'Banti's disease' had to be diagnosed. I also take this standpoint, and am more and more convinced that etiologically widely different chronic inflammatory processes of the



portal vein produce the picture and secondarily produce a sclerosis of the spleen in itself not characteristic."

Whichever way we turn we seem to meet with more confusion. Banti, from the first, contended that the disease in question had a characteristic pathology; it is obvious that his contention has been vigorously contradicted. Naegeli says that among German authors only Senator, Grawitz, and Umber agree with the ideas of Banti. The pathology as usually described is that of a diffuse perisplenitis and splenic fibrosis, with consequent destruction of splenic tissue.

The denial of characteristic pathology leaves in almost hopeless confusion any discussion of etiology and pathogenesis. While we dispute about the resulting pathology, whether it be primary and characteristic or secondary, the result of diverse diseases, our theories regarding the causation of the disease and its development must be widely variant. Does the condition result from the action of a single toxin which has a specific effect on the spleen? May a group of toxins acting simultaneously have a similar specific effect? May the same underlying factor cause atrophic cirrhosis if the liver is more susceptible to irritation, Banti's disease if the spleen has the lesser resistance? May toxins of widely different origin produce pathologic and symptomatic results, so much alike as to present the picture of a single disease? Is the term "Banti's disease" justifiable from any standpoint? Is it appropriate to any disease entity? If so, what is it, what causes it, and how shall we distinguish it? Shall the term be discarded because it is too inclusive, serving to mislead us into a careless acceptance of a vague term which serves to conceal ignorance? Must we look for an eventual reclassification of splenic cirrhoses on the basis of etiology?

These questions must be left unanswered. Meanwhile, however clouded the descriptive term "Banti's disease" may be from the theoretic or scientific standpoint, it still has a claim on our interest from the practical standpoint. As a well-established clinical entity we may refuse a place to "Banti's disease"; yet from the pragmatic standpoint, as a symptom-complex with its own problems of therapy, its own implications regarding prog-



nosis, the condition described by Banti still deserves consideration. How shall we classify the case before us if we can no longer speak of splenic anemia or Banti's disease?

Whatever we may think about nomenclature, or the further study necessary to answer the questions which have been raised, we believe that at present clinical experience still justifies us in the recognition of the symptom complex described by Banti as a practical clinical entity. The term includes a group of cases which cannot be classified in any other way. Splenic anemia and Banti's disease may be used as synonymous terms; they may be used to define different stages of the same disease; they can hardly be regarded as totally unrelated entities. Our views regarding the relationship of splenic anemia to Banti's disease would have only academic interest except for the fact that the disease is amenable in many instances to surgical treatment, if surgical treatment is employed prior to the development of hepatic disease. Splenectomy is the only therapeutic measure that has stayed the progress of this disease; it has been employed for many years, on the whole has given favorable results, and should be regarded as the indicated procedure. W. J. Mayo, who regards splenic anemia as a clinical entity, has recently discussed this subject. He refers to 74 operations for splenic anemia of unknown origin, with 9 deaths. While recognizing the difficulties of the operation, due especially to the tendency of the enlarged fibrotic spleens to become adherent, he regards his operative results as satisfactory.

And the terminology of the disease is of importance in reference to treatment. Splenectomy may be done with the greatest promise of success before the appearance of signs or symptoms of hepatic cirrhosis. Because this terminal stage is sometimes incorrectly spoken of as Banti's disease, it must be emphasized that the favorable time for operation is the stage of splenic anemia, the second stage of Banti's disease (as described by some), at which time there is splenomegaly with secondary anemia, without evidence of hepatic involvement. The sequence of events must be regarded; in the presence of a splenic anemia the subsequent hepatic involvement should be anticipated.

Timeliness of the operation is almost an essential to success, though W. J. Mayo has seen good results follow splenectomy "even in the terminal stage of the disease."

In closing may we briefly review the salient points of the clinic:

(1) A case has been presented showing splenic enlargement, secondary anemia of moderate degree, leukopenia, hepatic en-

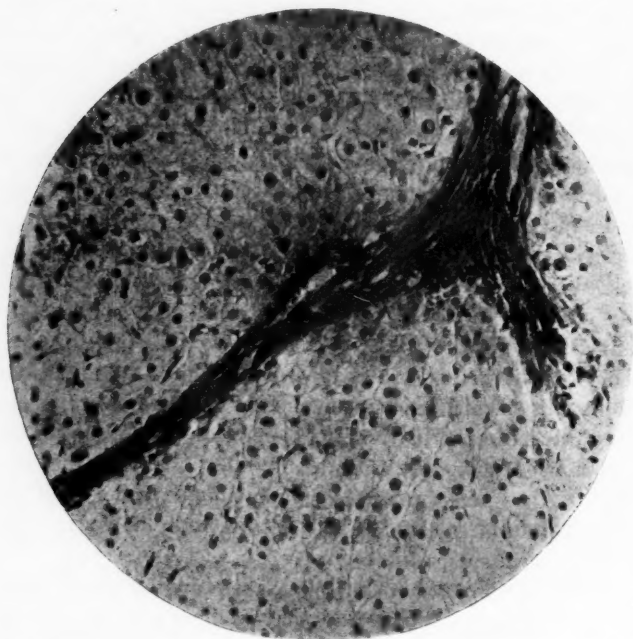


Fig. 295.—Section of the liver, showing a rather definite cirrhosis of the liver with changes in the structure and staining properties of the liver cells.

largement, and hematemesis. The disease has lasted over eighteen months. There is no evidence for the existence of syphilis.

(2) On the basis of these findings, and in the absence of specific etiology, the case is diagnosed as one of Banti's disease.

(3) A discussion of the subject of Banti's disease brought us

to the conclusion that, irrespective of the gaps in our knowledge, we are still warranted in regarding Banti's disease as a clinical entity.

(4) Splenectomy is advised and emphasis is laid on the value of the procedure if done before hepatic involvement is well established.



Fig. 296.—Section of the spleen. There is a fibrous tissue increase; obliteration of the lymph spaces, with a number of localized collections of round cells.

**Subsequent History.**—On October 27th a splenectomy was performed by Dr. W. R. Cubbins. The patient made an uneventful recovery.

The pathologic report on the removed spleen is as follows:

"This recently removed spleen weighs 520 gm. and is 18.5 cm. long, 10 cm. wide, and 6.8 cm. through. There are no very

marked changes in the external appearance except the large size, and there are in some areas a few small petechial hemorrhages into the capsule, and pinpoint white markings may be seen through the capsule. . . . Microscopic examination of sections from the liver and spleen reveals a rather definite cirrhosis of the liver, with changes in the structure and staining properties of the liver cells. In the spleen there is a fibrous tissue increase, obliteration of the lymph spaces, and a number of localized collections of round cells. Diagnosis: Banti's syndrome" (Figs. 295, 296).

## BLOOD COUNT

	Hb.	R. B. C.	W. B. C.
Two days after splenectomy . . . . .	56	3,970,000	18,700
Three days after splenectomy . . . . .	54	3,630,000	9,900
Five days after splenectomy . . . . .	50	4,160,000	12,800
Twelve days after splenectomy . . . . .	50	4,470,000	13,000
January 6, 1922 . . . . .	72	4,250,000	7,000
February 9, 1922 . . . . .	90	4,600,000	7,600

The counts made January 6th and February 9th showed 61 and 56 per cent. of polymorphonuclears respectively; the earlier counts showed much higher proportions of polymorphonuclears.

February 9, 1922 patient has gained 21 pounds since his operation; except for slight pain about the scar of the operation feels well, and would like to go to work.

Blood count, February 23, 1922: R. B. C. 5,000,000, W. B. C. 7000, Hb. 88.

## LEUKEMIA WITH GOUT AND HERPES ZOSTER

**Presentation of a Case of Splenomyelogenous Leukemia Complicated by Gout and Herpes Zoster. Therapy of Splenomyelogenous Leukemia. Relation of Leukemia to Herpes.**

WITHOUT undertaking any extensive discussion of leukemia we want to present to you a case of the splenomyelogenous type in which some interesting complications have developed.

J. E., age fifty, was admitted to our service August 16, 1921. About ten weeks prior to entrance he noticed swelling of the hands and feet, worse in the afternoon. About the same time he became aware of a dull, persistent pain in the legs, of such severity as to make walking difficult. Soon thereafter he discovered a swelling in the left hypochondriac region; this has grown steadily; it is not tender and is painful only when he attempts to work or to perform more than the lightest exercise. There are also present now "lumps" as large as walnuts over both elbows; the cause of their development is not well described; these tumefactions are freely movable and are not painful.

His appetite has been very poor. There has been moderate weakness, growing gradually worse. During the past four weeks there have been many attacks of dizziness; these are especially provoked by exertion. During the entire ten weeks of indisposition he has been unable to work. There is little of moment in his past history. In childhood he had measles and scarlet fever; three years ago he had influenza. Venereal disease is denied. His habits are good; he has been a steady worker, and uses neither tobacco nor alcohol. The family history is unimportant.

**Physical Examination.**—A poorly nourished man, rather underdeveloped, distinctly weak and anemic. Pulse 96, respirations 28, temperature 101.6° F., blood-pressure 125/55. There

is definite lymphadenopathy in the cervical, epitrochlear, and inguinal regions. The pupils are equal, regular, and react normally. The reflexes are normally active and no abnormal reflexes are present. The teeth are in fair condition, though they need some attention. The tonsils are fairly large, but free from evident disease. Pain was produced by movement of either ankle or the right knee. Many of the phalangeal joints of the right hand were swollen and stiff. Small hard granules could be felt in the lobes of the ears; according to the patient's statement these had first appeared after his ears were frozen.

The spleen was found to be greatly enlarged, extending as far as the left anterior superior iliac spine, while posteriorly it filled the lumbar region. The surface was smooth; palpation elicited no tenderness. The liver and kidneys were not palpable. Examination of the genitalia disclosed no abnormalities. Cardiac examination was negative. The lower border of pulmonary resonance was noted as high on the left side, and this finding was attributed to upward displacement of the lung and diaphragm by the enlarged spleen. Otherwise physical examination of the lungs gave normal results. The "lumps" over the elbows, already mentioned in the patient's description of his symptoms, were found to be soft, smooth, movable, and not painful, probably chronic inflammatory affections of bursæ.

Examination of the blood gave these results: Hemoglobin 38 per cent., red blood-cells 2,400,000, white blood-cells 500,000. A differential white count showed large and small mononuclears (total of the two varieties) 5 per cent., polymorphonuclears 45 per cent., basophils 20 per cent., transitionals 20 per cent., and myelocytes 10 per cent. Microcytes and macrocytes were present and poikilocytosis was pronounced. The urine was normal. Chemical examination gave per 100 c.c. of blood the following figures in milligrams: urea nitrogen 21.24, urea 45.39, uric acid 3.21, creatinin 1.46.

Fowler's solution was given in 7-minim doses t. i. d. for one week and then stopped. Benzol was administered with equal parts of olive oil in a dosage of 7 minims twice a day; after three days this was increased to three doses a day, and the daily dose

was increased 1 minim a day. When the benzol was stopped (the circumstances of which will soon be discussed) the patient was receiving 34 minims a day, in two doses of 11 minims and one of 12.  $\alpha$ -Ray therapy was begun on the day after admission. Five days after entrance, at which time the blood-count quoted above was made, and four days after the first  $\alpha$ -ray treatment, the total white count was only 282,500; four days later it was 147,500. Three Roentgen-ray treatments were given at intervals of one week, the last being given on September 2d. The total white count on September 5th was 20,450. The benzol was not given after September 2d. Without the continuance of these therapeutic measures the white count steadily declined; on September 22d the total count was 5150. Twice subsequently were the counts higher, but on October 7th the count was 4200; of these, the small mononuclears were 22 per cent., large mononuclears 4 per cent., polymorphonuclears 72 per cent., myelocytes 2 per cent. Coincident with the improvement in the white count the spleen became much smaller; the red count remained practically unchanged.

Just a few words regarding the therapy of myelogenous leukemia before we take up those features of the case which will more especially engage our attention. On the basis of our own experience Roentgen-ray therapy prolongs the life of the individual afflicted with leukemia. Often this form of treatment adds a few comfortable years to the patient's life; I have seen no cures. The time does come, somewhat later with the use of the  $\alpha$ -ray than without, it is true, but the time does come when treatment no longer has any inhibiting effect on the progress of the disease.

Benzol will reduce the number of leukocytes, but its use is not unattended with danger. This danger is the result of the action of benzol on the blood-forming organs, and may announce itself by (1) a continued decrease of the leukocytic elements in spite of the withdrawal of the drug, (2) a rapid decrease of the red cells. In other words, under benzol therapy an aplastic anemia of uncontrollable nature may replace the original leukemia. It is this feature of benzol activity which is to be



feared. During its administration careful study of the blood-picture is imperative. Here we stopped the benzol when the total leukocyte count fell to 25,000; no x-ray exposures were given after the count was 20,000; yet the decline in the leukocyte count continued to such an extent (4200 at the lowest) as to cause us some anxiety.

Upon admission the temperature was 101.6° F.; the temperature became normal the first day after entrance, and remained so throughout the first week. On August 24th there was a rise of temperature to 100.6° F., accompanied by pain, swelling, and tenderness of the right great toe. These phenomena considered in connection with the hard granules in the ears (now believed to be tophi), with the involvement of several phalangeal articulations and the high uric acid content of the blood, led us to the diagnosis of gout. Not infrequently gout is a complication of leukemia; especially is an acute exacerbation likely to occur with the rapid destruction of leukocytes, which so often follows the use of Roentgen therapy. The nucleoproteins of the leukocytes form the material which is elaborated into uric acid; thus we can account for the high blood uric acid discovered even before the beginning of treatment. I regret that later studies were not made. Late in September another acute exacerbation occurred, less severe in its manifestations; in this attack both great toes were mildly involved. Atophan gave prompt relief in both of these acute attacks.

For a few days prior to September 10th the patient complained of pain in the left shoulder, about the point of the shoulder, and along the posterior aspect; the pain really began not far from the midline and followed a line along the suprascapular region, past the point of the shoulder, down the outer aspect of the arm. It was not associated with any evidence of disease of the joint. Tenderness along the course described was marked. On September 10th it was noted that "Small groups of papulovesicular lesions are present over the skin of the left arm (Fig. 297). There is pain in the left chest. The temperature is 100° F. There is no evidence of pulmonary disease." Two days later this notation occurs, "Skin lesions are confined to the



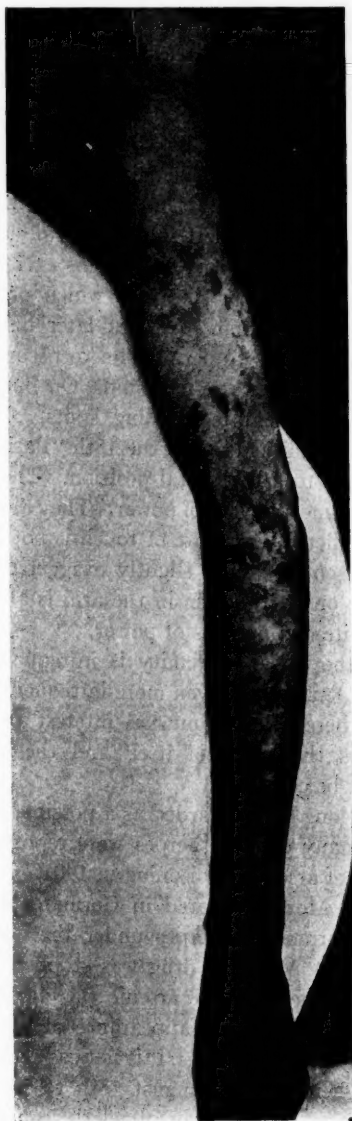


Fig. 297.—Photograph showing the herpetic lesions on the left arm.

left arm, causing burning and pain. The condition is regarded as a herpes." On the 14th "Great pain in the left arm. Lesions show hemorrhages into the larger ones." You observe today, a month or more since these notations were made, the crusted areas and the scars, principally confined to the outer aspect of the left arm and forearm. The pain did not disappear with the subsidence of the eruption. On September 28th the record shows that the pain in the left shoulder, arm, and hand was so severe as to interfere with the use of this extremity. The patient described the pain as like that which preceded the first appearance of the eruption. A week later his general condition was so improved as to permit him to be up and about the ward; the pain in the left arm persisted. On October 12th "The left arm shows pigmentated scaling spots. Pain continues. Power is much diminished." On the 18th "Patient is unable to raise the left arm to a horizontal position. The fingers can be moved, but are almost devoid of power. They are shiny, smooth, and slightly swollen. The sense of touch is not impaired. The reflexes are present, possibly slightly exaggerated." Now, six weeks after the onset, the pain in the arm is at times extreme, and there is quite distinct loss of power.

Herpes of the upper extremity is infrequent; herpes as a complication of leukemia is even more infrequent. Thus it happens that the herpes lends unusual interest to the case. As etiologic factors the leukemia itself and the arsenic used in treatment must be considered.

Arsenic has long been regarded as a possible cause of herpes. For instance, many cases of herpes were reported in the Midlands outbreak of arsenical poisoning in 1901. Not long since a contribution by Mezei appeared in Germany in which he remarked the occurrence of herpes under the administration of Fowler's solution. After the drug was stopped the herpes disappeared; some time later the arsenic was again administered, only to be followed by the reappearance of herpes. Granting that arsenic may produce herpes, it is not easy to accept it as the effective cause here. From August 18th to 23d inclusive this patient was given 2 drams of Fowler's solution; beginning

September 3d it was again given, this time for nine days, a total of 3 drams being given. The dose employed through each period was 7 minims three times a day. It is not likely that a total of 5 drams of Fowler's solution, distributed in its administration over a period of twenty-four days, could cause a herpes. The loss of power noted in the record might suggest a complicating neuritis and thus make more plausible the view that the arsenic was the offending agent. Arsenic would be more likely to cause a complicating neuritis, in addition to the disease of the root ganglia, than would leukemia. The presence of the reflexes and the lack of marked atrophy are difficult to reconcile with a diagnosis of neuritis; the loss of power may be the result of the constant pain; for weeks the left arm has been carefully kept quiet to avoid pain. Finally, some loss of power has often been noted in association with herpes.

Has the leukemia an etiologic relationship to the herpes? Speculation is easy; proof is difficult. In the literature there are few references to herpes as a complication of leukemia. Leukemic infiltration of the spinal cord has been described. Not long since Basso described a case of paraplegia complicating leukemia; the paraplegia was "caused by purely mechanical compression of the cord by a leukemic tumor growing into the spinal canal." Basso refers to the collection by Baudoin and Parteurier of 26 cases of involvement of the nervous system in leukemia including 7 of leukemic infiltration of the cord or brain. Prior to Basso's case 2 cases of paraplegia due to leukemic infiltration of the spinal dura had been reported; of these, one case had been called in question. The other case was that of Stursberg. This case of Stursberg is the only one mentioned by Naegeli in his reference to the subject of leukemic involvement of the spinal cord.

Herpes was recently listed among the nervous complications of leukemia by Murray Bass; this classification was taken from an article in the French literature, and Bass made no reference to the occurrence of herpes in any of his own cases.

Dr. Pollock has suggested the possibility of leukemic infiltration of the posterior root ganglia to account for the case of

herpes before you. We are not convinced of the etiologic relationship of the Fowler's solution; the suggestion regarding leukemic infiltration is interesting and plausible. A final decision or a positive assertion as to the cause of this herpes cannot be offered to you now. It may have been only an incidental intercurrent herpes.

## CLINIC OF DR. CHARLES A. ELLIOTT

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, WESLEY MEMORIAL  
HOSPITAL

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### CLINICAL TYPES OF GOITER AND THEIR MANAGEMENT

**Our Present Conception of the Treatment of Various Types of Goiter and Particularly of Hyperthyroidism. Classification of Goiters. Differential Diagnosis of Hyperthyroidism. Basal Metabolic Rate. Therapeutic Measures. Presentation of 9 Patients with Different Types of Goiter.**

THIS clinic will be devoted to a discussion of our present conception of the treatment of the various types of goiter and particularly of hyperthyroidism; to an evaluation of the various therapeutic agents at our disposal, and to the presentation of patients and abstracts of case records illustrating the results of treatment.

It is necessary first to understand exactly what is meant by the terms applied to the various types of goiter. We have adopted the following working classification which, it must be remembered, is more or less artificial (and can never be exact) because of the many variations and combinations of types and because of changes from one type to another in individual cases.

#### CLASSIFICATION OF GOITERS

1. **Simple goiter**, called by various names, endemic by Marine, is the goiter frequently seen in adolescence, pregnancy, and the menopause. These patients show only slight to moderate degrees of intoxication; they are rarely severely toxic. Symptoms are apparently due to physiologic overactivity of the gland.

Microscopically there is apparent hypertrophy of the normally secreting gland. The basal metabolic rate averages from 0 to +20.

**2. Goiter Clinically Non-toxic.**—This group comprises all of the large strumæ unassociated with hyperthyroidism. The enlargements may be cystic, colloid, adenomatous, or may show other changes under the microscope. They are often large, sometimes irregular. The only complaint is that of the tumor mass itself or of symptoms produced by pressure of the mass. Goiters of this type may at any time become toxic, as described in the following group. The basal metabolic rate is within normal limits, *i. e.*, -10 to +10.

**3. Toxic Goiter.**—This group includes all of the obviously toxic types of goiter save true exophthalmic goiter. It is a large and heterogeneous group often called secondarily toxic goiter, pseudo-Graves' disease, atypical hyperthyroidism, adenoma with hyperthyroidism, and other names applied to more specific subdivisions descriptive, usually, of the histologic findings thought to be present. It seems to us, from a clinical standpoint, that a greater subdivision of this group leads only to confusion. These goiters are of long standing; they may be of various types—simple, colloid, cystic, adenomatous, or even malignant. Symptoms may have existed for a long time, but usually show marked exacerbation just before the patient applies for medical relief. It is, therefore, considered secondarily toxic. The course is usually indefinite with irregular periods of intoxication.

Exophthalmos and gastro-intestinal crises are rare. Hypertension may be present. The basal metabolic rate ranges from +30 to +60.

**4. Exophthalmic goiter** (true Graves' disease or Basedow's disease) is characterized by hyperplasia of the parenchymatous cells. The thyroid gland may or may not be enlarged. The clinical course is definite and progressive, although with remissions and exacerbations. Exophthalmos, gastro-intestinal crises, and an intense general intoxication are usually present. The basal metabolic rate ranges from +33 to +170.

**DIFFERENTIAL DIAGNOSIS OF HYPERTHYROIDISM**

Because of the necessity of early diagnosis and treatment of exophthalmic goiter it may be well to indicate some points of difference between this type of goiter and secondarily toxic forms, particularly the adenoma with hyperthyroidism described by Dr. Henry Plummer.

In **exophthalmic goiter** the course of the disease is usually continuous and definite. The onset is rather acute. The thyroid gland itself may or may not be enlarged; if enlarged, the symptoms develop with the enlargement. The gland is uniform and symmetric. The superior thyroid arteries are said to be palpable in most cases. A thrill and bruit are usually present; one hesitates to make a diagnosis of exophthalmic goiter in the absence of a bruit. There is a definite fine tremor. An early tachycardia, 15 to 20 beats more than would be expected from the temperature, persists under all conditions. There is no tendency to hypertension; diastolic pressure is low, pulse pressure high. Gastro-intestinal crises are frequent. Basal metabolic rates, in our experience, vary from +55 to +170.

In **adenoma with hyperthyroidism** the onset is insidious and the course is variable, often extending over many years. Enlargement of the thyroid gland has usually been present for years; there may be a history of repeated periods of hyperthyroidism during this period. A thrill and bruit are almost uniformly absent. Tremor is irregular and less marked. Exophthalmos rarely, if ever, occurs. There is a definite tendency to hypertension; the diastolic pressure is sustained and the pulse-pressure is normal. The basal metabolic rate, in our experience, has varied from +30 to +60.

Patients with toxic adenoma or other less clearly defined secondary hyperthyroidism are apt to seek relief from grave cardiac manifestations. These are, of course, advanced cases in which the heart condition so overshadows the evidence of existing or previous hyperthyroidism that a proper interpretation of the former manifestation is often difficult.

Occasionally cases of exophthalmic goiter or of secondary hyperthyroidism resemble clinically certain neuroses, especially

the so-called effort syndrome, and certain infections, particularly early pulmonary tuberculosis. Metabolic rate determinations have been of the greatest value to us in ruling out these conditions. In exophthalmic goiter at this stage the metabolic rate usually varies between +30 and +80, while both in the neuroses and in tuberculosis the rate is not far from normal. In hyperthyroidism the tachycardia is persistent; in effort syndrome it is aggravated by physical effort and in tuberculosis it is variable. A bruit heard over a thyroid gland which may be only very slightly enlarged is a valuable sign suggestive of hyperthyroidism.

#### BASAL METABOLIC RATE

The basal metabolic rate determination has been used as an aid in diagnosis and as an index to the effect of treatment. It has proved the best single guide in both of these instances. It is not, however, in our experience always an accurate guide as to the degree of thyroid activity. Some patients are manifestly much more toxic than would be indicated by their metabolic rate. There are many conditions other than toxic goiter in which the metabolic rate is higher than normal; for example, patients seriously ill with carcinoma, heart and kidney diseases, and infections. In febrile conditions the metabolic rate is usually increased, often comparable to that of a moderately severe hyperthyroidism. In these cases there is usually no question as to diagnosis. The following table shows the minimum and maximum rates obtained in various types of goiter and in other common clinical conditions seen in our clinic:

#### BASAL METABOLIC RATES IN THYROID CONDITIONS

Exophthalmic goiter.....	+33 to +170
Toxic goiter.....	+19 to +60
Goiter of adolescence.....	+10 to +20
Goiter of pregnancy.....	+ 4 to +32
Goiter of menopause.....	+12 to +28
Cretinism.....	-10
Myxedema.....	-9 to -14



## BASAL METABOLIC RATES IN OTHER CLINICAL CONDITIONS

Patients not seriously ill.....	-4 to +22
Patients seriously ill (cancer, heart and kidney disease, high fever).....	+20 to +60
Focal infections.....	-15 to +30
Neuroses.....	-14 to +29
Pulmonary tuberculosis.....	+8 to +33
Prolonged inanition.....	-37 to -10
Diabetes (severe).....	+10 to +33
Diabetes (emaciated).....	-35 to -10
Obesity.....	-2 to -9
Addison's disease.....	-29
Osteomalacia.....	+7

The metabolic rate often varies greatly from day to day in individual cases. The first determination is, in our experience, apt to be fallacious, due probably to the discomfort of the mask and the apprehension of the patient as to the significance of the procedure. The following table illustrates the variations that may be seen in individual cases and the results of various forms of treatment, including x-ray, radium, removal of foci of infection, and subtotal thyroidectomy.

## BASAL METABOLIC RATES—INDIVIDUAL CASES

1. *Exophthalmic goiter* (Mrs. McE.):
 

3/23/21.....	+33
3/27/21—subtotal thyroidectomy	
5/10/21.....	0
8/22/21.....	-5
2. *Exophthalmic goiter* (Miss H., twelve years):
 

11/2/20.....	+58
11/30/20—after six x-ray treatments.....	+60
1/5/21—after twelve x-ray treatments.....	+48
1/6/21—subtotal thyroidectomy	
2/17/21.....	+1
3. *Exophthalmic goiter* (Mrs. R.):
 

12/11/21.....	+154
12/17/21—1200 mg. hours radium	
12/20/21.....	+95
12/28/21—1560 mg. hours radium	
1/3/22.....	+170
2/1/22—death	

4. <i>Toxic adenoma</i> (Mrs. Van S.):	
11/20/20.....	+55
12/10/20.....	+66
12/15/20—subtotal thyroidectomy	
2/7/21.....	-9
5. <i>Toxic adenoma</i> (Mr. W.):	
8/4/21.....	+60
8/9/21—subtotal thyroidectomy	
11/5/21—after tonsillitis.....	+31
11/25/21.....	+39
12/4/21—tonsillectomy	
12/20/21.....	+40

### TREATMENT

In order to systematize our ideas it may be well first to list the various agents available in the treatment of hyperthyroidism and to discuss their relative values.

**Therapeutic Measures.**—1. Early diagnosis and classification of cases for the purpose of prompt operative interference, where indicated, are of such great importance as to warrant consideration as the most urgent therapeutic indication. The parenchymatous changes resulting from hyperthyroidism are more or less permanent. Early detection and control are necessary to insure permanent cure and a minimum of tissue changes.

2. *Rest in bed* has been recognized for many years as a useful agent in the treatment of hyperthyroidism. With some it has been the sole agent employed. Many clinicians have been in the habit of keeping hyperthyroid patients in bed, often for months at a time. Bed rest is undoubtedly an important agent; in some cases it may be all that is necessary. In our experience rest in bed seems to act solely as a supporting agent, enabling the patient better to withstand the intoxication; it seems to have little or no influence on the production of thyroid secretion. The maximum benefit should usually be obtained after a very few days. Five to seven days' rest in bed, in the average case, seems to us sufficient before proceeding to more active treatment. There are, of course, exceptions to this rule: patients desperately ill, poor operative risks, patients with

auricular fibrillation, mental hyperexcitability, and those with marked vasomotor irritability should be kept in bed to await a favorable opportunity for operative interference.

It is of distinct advantage to encourage patients in whom the diagnosis is still in doubt to follow their ordinary routine. Being up and about, getting physically tired or out of patience (conditions which accentuate the symptoms of hyperthyroidism) are of advantage in making possible an accurate diagnosis in some of the borderline cases.

3. *Iodin*.—We have followed the teaching of Marine regarding the use of iodine in the treatment of hyperthyroidism. It seems apparent that when the iodine store in the circulating blood is low the thyroid is stimulated to increased activity. Maintaining the iodine store of the blood has seemed to give a definite protection against compensatory hypertrophy and hyperplasia of the thyroid gland. Under the influence of iodine intoxication seems to be reduced and the size of the gland decreased. The administration of thyroid substance is said to produce much the same result; we have had no experience with it in hyperthyroidism. We have been careful to administer iodine to patients with hyperthyroidism only where they have been under continuous clinical supervision; no ill effects have been observed from its use. We have used iodine in various forms, more recently as sodium iodide, about 2 grams a week. In our experience the form in which iodine was administered made little or no difference in the results.

It seems certain that the use of iodine in the milder secondarily toxic goiters and especially in the simple goiters of puberty, pregnancy, and the menopause has been of great value; in the more severely toxic adenomas it has occasionally seemed beneficial; and in the true exophthalmic goiter it has been of no value.

4. *The Removal of Focal Infections*.—Our experience in this regard has left us with the conviction that infection of any kind—general, local, or focal—has a profound influence on the activity of the thyroid gland. The frequency with which thyroid overactivity has occurred during the convalescence or has

followed in the train of general infections, such as influenza, bronchopneumonia, acute tonsillitis, etc., and the frequency with which thyroid overactivity has seemed to be reduced by the removal of evident foci of infection, especially those about the head (regional), have convinced us that an important relationship exists between infections and thyroid activity. The nature of this relationship is not entirely clear; it may be a direct result of the action of the organisms on the thyroid itself, or it may be concerned with a more indirect effect on the chemistry of the body, such as an influence on the iodine store of the blood.

Manifest foci of infections should be removed for the specific purpose of influencing thyroid activity as well as from the standpoint of general health insurance despite prejudice aroused by the wholesale removal of doubtful foci. In severe hyperthyroid cases removal of foci of infection should follow the more active measures, such as operative procedures directed against the source of toxemia; but in the milder forms of hyperthyroidism they may be treated at the start. In some of our patients the removal of manifest foci of infection has apparently been all that was necessary. It seems manifestly wrong to subject an intensely toxic patient to the removal of foci of infection, especially when these are doubtful, without having first controlled the hyperthyroidism as by operative interference. In such a case the time element is of the greatest importance; the patient is constantly receiving doses of thyroid secretion sufficient to produce progressive parenchymatous damage. The amount of tissue degeneration that may occur in a single day is not negligible. It is, therefore, advisable to attack the hyperthyroidism directly and, particularly in the more severe cases, without delay.

5. *Radium and x-Ray.*—The use of radium and x-ray as methods of control of severe hyperthyroidism has been a disappointment to us. It may be that we have not been in a position to apply them as they should be applied. Both undoubtedly exert a definite influence on the activity of the thyroid; in some of our patients their use preliminary to operative interference

has been of decided benefit. We have not observed the definite and lasting results which would stamp these agents as all sufficient in the treatment of hyperthyroidism.

There are several difficulties encountered in the therapeutic use of radium and x-ray in hyperthyroidism; of these, the following may be mentioned: difficulty in regulating the dose; the necessity of taking into consideration the effect upon the parathyroids and thymus; changes in surrounding connective tissue which may make later operative interference difficult.

In general, it must be admitted that radium and x-ray therapy may be sufficient in any given case and that they are useful in reducing hyperthyroidism preliminary to operative interference.

6. *Operative interference*, in our experience, has proved to be the most effective means of combating hyperthyroidism. It must be admitted, however, that only in certain cases and under certain conditions does it prove all sufficient. The results necessarily vary with the time and type of operation as adapted to the individual patient.

Any procedure which causes destruction of thyroid cells will reduce hyperthyroidism. For example, *ligation of the thyroid vessels* produces atrophy of the gland and may be a sufficient measure. We have used it chiefly as a step preliminary to final control by thyroidectomy. *Lobectomy*, as performed commonly until about three years ago, has, for the most part, been discarded as unsatisfactory because of the incomplete results obtained and because of frequent immediate and severe postoperative reactions. The severe toxic reaction was conceivably due to traumatism or to other postoperative overstimulation of the remaining lobe. There seemed, moreover, to be a predisposition to later exacerbations. Accordingly, the amount of thyroid substance allowed to remain and not the amount of thyroid tissue removed seems to us the dangerous factor. Since our surgical colleagues have ventured to remove much more of the gland substance there have been fewer immediate serious reactions and fewer cases requiring further operations.

*Subtotal thyroidectomy*, the removal of practically all of the thyroid substance except a thin sheet at the back of the gland to protect the parathyroids, has proved to be the optimal procedure for exophthalmic and other severe toxic goiters. The very small amount of gland left *in situ* has proved sufficient to perform the necessary thyroid function. Following subtotal thyroidectomy we have seen prompt and satisfactory results in most cases. Where the intoxication has not been allowed to continue over a long time and where, as a result, severe parenchymatous changes have been avoided (*i. e.*, when an early diagnosis was made and a subtotal thyroidectomy performed) a prompt and apparently a complete cure has resulted. After the operation the intoxication has been observed to disappear rapidly, sometimes within a few hours; the basal metabolic rate has fallen to within normal limits, and but few of the severe immediate reactions so often developing after lobectomy have occurred.

#### PLAN OF MANAGEMENT OF THE DIFFERENT TYPES OF GOITER

Among the most important of the many factors to be considered in the treatment of goiter are the great variety of clinical types which occur and the extreme variability in the degree of intoxication that exists in individual cases. Save in exophthalmic goiter, the variable intensity and duration of intoxication both in the groups described and in individual cases make the treatment always an individual matter. We have found it desirable, nevertheless, to formulate a general plan of procedure for each of the main groups of the disease. The details are varied to meet individual requirements.

1. **Simple Goiter.**—In the management of these cases the prophylactic treatment of school children living in endemic centers, as followed by Marine, should first be mentioned. One or 2 grams of sodium iodid, in small doses over a period of a month twice each year in adolescents from eleven to seventeen years of age, has been demonstrated to be effectual.

Iodin in any form in moderate sized doses, given before and during menstruation, pregnancy, or the menopause has

proved of definite value in reducing intoxication and attendant symptoms.

No further management is necessary in the majority of cases, although foci of infection should, in our opinion, be eliminated, and meat in the diet should probably be restricted, as suggested by the work of Bensley.

2. **Goiter clinically non-toxic** should be treated solely on the basis of the tumor; if large enough to produce local pressure on neighboring organs it should be removed. Removal for cosmetic effect alone seems justifiable.

3. **Toxic goiter** requires more ingenuity and judgment in treatment than any other type of goiter. Treatment varies according to the degree of hyperthyroidism. *Mild cases* have been given moderate doses of sodium iodid, 8 to 10 grains, three times daily, and, in addition, any foci of infection present, especially about the head, have been removed as soon as possible. The effect has usually been definite, and in some cases marked and sufficient. A number of such patients under prolonged observation have apparently been cured by the removal of foci of infection, but such happy results are only anticipated in exceptional cases. Roentgen rays, radium, ligation, or the enucleation of adenomata are indicated in selected cases and sometimes prove sufficient. If there is no response to the above measures, *i. e.*, if hyperthyroidism continues, a subtotal thyroidectomy should be done. In *severe cases* the same procedure outlined for exophthalmic goiter is followed.

A large percentage of the patients in this group complain of cardiac symptoms which have become rather suddenly severe. On examination a well-developed thyroid heart or auricular fibrillation, or both may be found. These are apt to be cases of adenomatous goiter. The cardiac condition may demand immediate attention, bed rest, and digitalization. The usual beneficial effects of digitalis are less likely to be obtained than in comparable cardiac conditions due to other causes. Morphin or powdered opium have been useful. Morphin and digitalis are used routinely as preoperative and postoperative measures.



4. **Exophthalmic Goiter.**—We are convinced that in this type of hyperthyroidism early subtotal thyroidectomy is indicated in all cases. Early diagnosis and operation will produce a complete and satisfactory cure in the great majority of cases; operation performed after a more prolonged period of intoxication produces results directly proportional to the extent of parenchymatous changes that have taken place. The earlier the operation, the less is the danger and the more likelihood is there of complete recovery. In our experience the operative risk is slight in early cases; there is also little danger of immediate postoperative reactions. Roentgen ray or radium treatment or ligation may be advisable in some of the more severe cases preliminary to subtotal thyroidectomy. These are the more severely toxic patients who apply for relief late in the course of the disease; early cases require no such preliminary measures.

All patients are kept at rest in bed for relatively few days prior to operation; five to seven days is usually sufficient. During the convalescence from operation the patients should be examined especially for foci of infection, which should be removed when present.

The management of patients in whom operation is contraindicated because of the presence of excessive toxemia, effects of hyperthyroidism, or associated pathology has proved very unsatisfactory in our hands. The outlook for such patients is grave; they either die or are permanently incapacitated. Patients showing marked mental symptoms, gross vasomotor manifestations, extreme nervousness, and auricular fibrillation are considered bad operative risks, even in the presence of metabolic rates which are not excessive.

In a few patients it has seemed impossible to make a diagnosis of early exophthalmic goiter with certainty. Subtotal thyroidectomy performed in a few such cases in which the diagnosis seemed reasonably certain proved beneficial, and in no case has the procedure been regretted.



## PRESENTATION OF CASES

CASE I (No. 94,217).—Mrs. A. S., forty-two years of age, entered the hospital August 22, 1921. She had noticed symptoms for two months, viz., loss of strength, palpitation, dyspnea on exertion, nervousness, sleeplessness, abdominal pains, nausea, and loose bowel movements. Enlargement of the thyroid gland, tremor, and exophthalmos had only been noticed for a few days.

On physical examination the patient appeared nervous; eyes were somewhat prominent; the thyroid was uniformly but only moderately enlarged, a bruit was heard over the gland; the heart was normal but rapid, pulse 96 to 120, blood-pressure 130/76; slight ankle edema was present; the urine showed a trace of albumin, no casts; red blood corpuscles 4,032,000, white blood corpuscles 7400, hgb. 90 per cent.; Wassermann test negative; basal metabolic rate +17.

A diagnosis of *beginning exophthalmic goiter* was made, and a subtotal thyroidectomy was performed by Dr. Harry M. Richter on August 27, 1921. Recovery was rapid and apparently complete. The patient left the hospital on September 10, 1921.

On January 26, 1922 the patient returned for examination, and stated that she felt perfectly well and had gained 20 pounds in weight. All of the symptoms of which she had complained on admission had disappeared. On physical examination there were no evident manifestations of hyperthyroidism. The basal metabolic rate was +2.

This case illustrates the prompt and complete results that may be obtained by subtotal thyroidectomy when performed early in exophthalmic goiter (in this case two months after onset). In our experience there is little or no danger in the operative interference in these early cases—probably no more danger than operating upon non-toxic goiters. The results obtained are in great contrast to those which follow operation performed late, after permanent tissue damage has occurred. The latter results are incomplete and the patient is usually doomed to a life of invalidism.

CASE II (No. 89,640).—Mr. W. B., a department store buyer, thirty-four years of age; admitted December 10, 1920, with a history of goiter of seven months' standing, pressure symptoms, exophthalmos, loss in weight and strength, nervousness, tremor, tachycardia, palpitation, sweating, and recent edema of the feet. He had had a partial thyroidectomy performed June 20, 1920, but the symptoms continued. Dyspnea and edema of the legs became prominent.

Physical examination revealed evidence of *well-advanced exophthalmic goiter with marked cardiorenal changes*. The heart area was broad, a systolic murmur present at the apex, pulse 114, regular; blood-pressure 170/72. Basal metabolic rate December 11th, +42. A subtotal thyroidectomy was performed December 19, 1920 by Dr. Richter. A large substernal portion of the thyroid was found and removed.

The patient improved promptly; the edema, tachycardia, and heart murmur disappeared. He returned to work in March, 1921 and has been able to perform his duties as well as ever. On March 4th the metabolic rate was +8; on December 16th, +14. A year after the final operation he had gained 33 pounds in weight. The heart remains definitely hypertrophied.

This is a case of *well-advanced exophthalmic goiter with visceral changes* and with cardiac decompensation. Partial thyroidectomy had produced no improvement, but a subtotal operation definitely checked the toxemia and permitted compensatory cardiac hypertrophy. A certain degree of myocardial damage seems to be permanent, but functional restitution is practically complete.

CASE III (No. 91,398).—Mrs. M. McE., forty-five years of age, was admitted March 22, 1921. In November, 1920 she had had a general infection, with fever, diagnosed by her family physician as influenza. She remained in bed for three weeks. During convalescence the thyroid gland seemed suddenly to enlarge, and symptoms of exophthalmic goiter rapidly developed. Four radium treatments were given by the family physician, one each month from November to February, but without evident effect.

On admission, four months after the onset of the hyperthyroidism, signs of *well-advanced exophthalmic goiter* were present; of these, exophthalmos, enlarged thyroid gland with bruit, and tachycardia were prominent. Basal metabolic rate March 23d was +33.5. On March 27th a subtotal thyroidectomy was performed by Dr. Allen B. Kanavel. The patient promptly recovered from the operation, but continued to complain of diminishing vision and of edema of the conjunctivæ and eyelids and exophthalmos. On August 22, 1921 the basal metabolic rate was -5. The patient was unable to read ordinary news print due to the presence of a marked neuroretinitis (Dr. E. V. L. Brown). The condition of the eyes gradually improved; by February, 1922 the swelling of the lids and conjunctivæ, exophthalmos, and the vision were all definitely better, but far from normal.

This case illustrates the apparent influence of a general infection on the thyroid gland (the development of hyperthyroidism during convalescence). Such relationship between general infections and hyperthyroidism seems not uncommon in our experience. It also illustrates the incomplete results obtained when operative interference is delayed until after extensive parenchymatous changes have occurred.

CASE IV (No. 96,264).—Mrs. D. R., forty-four years of age, entered the hospital December 16, 1921. Enlargement of the thyroid gland was first noticed in the spring of 1921; it was never marked. Exophthalmos, goiter with bruit, vasomotor instability, loss in weight and strength, nervousness, tremor, tachycardia, irregular pulse—150 to 180, palpitation, sweating, diarrhea, jaundice, and cervical adenitis were noted. Red blood-cells 3,948,000, white blood-cells 12,300, hgb. 85 per cent.; urine contained albumin and casts; Wassermann test negative; blood-pressure 138/72; basal metabolic rate +154.

Operation was considered contraindicated, and it was decided to try radium treatment. On December 17th 1200 mg. hours of radium element was applied over the thyroid gland. On December 20th the basal metabolic rate was +95. On December 26th the patient's condition was very grave; jaundice

was intense, liver enlarged, edema was marked in the legs and left arm. On December 28th 1560 mg. hours of radium element was applied over the thyroid. On January 3, 1922 the basal metabolic rate was +113. By January 18th the patient's condition was much worse; extensive edema of dependent parts, especially of the left arm, was present; jaundice was intense; basal metabolic rate +126. Death occurred on February 1, 1922.

This case illustrates the ineffectiveness of treatment in *far-advanced exophthalmic goiter*. Radium was of no value.

*Necropsy Findings* (Dr. James P. Simonds).—Exophthalmos; general icterus; edema of legs, thighs, left upper extremity, and of the tissues of the anterior mediastinum and left supraclavicular fossa; slight ascites. Thin scalp and scanty axillary and pubic hair. Atrophy of the mammary glands.

*Combined exophthalmic and adenomatous goiter*; cystic adenoma of the thyroid gland. Hypertrophy of the veins from and enlargement of the lymph-nodes regional to the thyroid gland. Persistent thymus.

Cardiac hypertrophy (moderate) and dilatation; petechial subpericardial hemorrhages; fatty degeneration of the myocardium; chronic mitral endocarditis; patent foramen ovale with fibrous cord across the opening in the right auricle which contained multiple mural thrombi. Thrombosis of the left innominate vein extending into the left jugular and subclavian veins with partial organization in the subclavian vein. Thrombosis of the branch of the right pulmonary artery to the lower lobe and of its intrapulmonary divisions. Slight atheroma of the aorta. Localized fibrinous pleurisy and multiple infarcts of the right lung with compression atelectasis produced by pleural exudate. Gangrene of the left lung; chronic obliterative pleurisy. Enlarged mediastinal and peribronchial lymph-glands. Chronic diffuse nephritis of parenchymatous type; anemic infarct of right kidney. Slight edema of the brain, with hyperemia of the vessels over the vertex. Moderate hypertrophy of the pineal gland and slight hypertrophy of the hypophysis. Marked fatty degeneration of the liver; chronic peri-

hepatitis; chronic pericholecystitis. Hyperemia of the pancreas; hypertrophy of the spleen; moderate hypertrophy of the adrenals with diminished yellow pigment in the cortex.

CASE V (No. 88,857).—B. H., a school girl twelve years of age. Enlargement of the thyroid gland was first noticed in April, 1920; exophthalmos was noted by the mother two months later; then loss in weight and strength, nervousness, tremor, tachycardia, palpitation, sweating, flushing, and headache. The child had had occasional sore throats and the tonsils had been removed.

On October 29, 1920 the pulse-rate was 140; temperature 99.4° F.; tremor, nervousness, exophthalmos, and general adenopathy were present. Part of one tonsil remained; ears and teeth were free from infection; lungs negative; heart rapid, area broad, no murmurs; thyroid diffusely enlarged with a loud bruit heard over the gland. Wassermann test negative; blood-pressure 134/60; basal metabolic rate +58.

On account of the age of the patient it was decided to avoid operative interference if possible. She was ordered to remain in bed. November 6th to 10th four Roentgen-ray exposures were made over the thyroid region. On November 30th her condition was unchanged; basal metabolic rate +60. A second series of Roentgen-ray exposures were made without apparent benefit. January 5, 1921 basal metabolic rate was +48. No improvement having been obtained after more than two months' rest in bed and after Roentgen therapy, surgical intervention was advised. Subtotal thyroidectomy was performed by Dr. Richter on January 6, 1921.

After the operation the symptoms of hyperthyroidism rapidly disappeared; by February 17th she had gained 12 pounds in weight; the heart appeared normal, rate 80; tremor and exophthalmos were no longer present; basal metabolic rate +1. She appeared to be in perfect health without evidence of previous hyperthyroidism, and has so continued.

This case illustrates the apparent fact that exophthalmic goiter may run the same course in children as in adults, and that the same plan of treatment may be followed in both.

CASE VI (No. 95,988).—Mrs. M. F., sixty-four years of age. Eleven years before admission to the hospital this patient had had a cholecystectomy performed because of gall-bladder disease. Shortly thereafter a large portion of a very large thyroid gland, thought to have been a colloid non-toxic goiter, was removed.

On admission, October 30, 1921, the complaint was that of progressive weakness, loss of 60 pounds in weight, depression, voracious appetite, insomnia, pain in the shoulder regions, frequent urinations, especially at night, dyspnea on effort. The patient stated that sugar had frequently been found in the urine. A diagnosis of diabetes had been made and a diet had been prescribed and followed for three years. The symptoms, however, continued and even became gradually more severe.

Examination revealed a stout woman whose flabby, pasty skin gave evidence of loss in weight. Ankles were slightly edematous. A large and irregular goiter without bruit which had apparently existed for years without change was present. The heart showed evident enlargement; no murmurs or irregularity. Blood-pressure 192/102; Wassermann test negative; basal metabolic rate +33. Later metabolic rates were +22 and +27 on November 14th and 21st.

On November 28, 1921 a subtotal thyroidectomy under local anesthesia was performed by Dr. Kanavel. Recovery was uneventful; rapid improvement in her general condition followed. Basal metabolic rate on December 14th was +9. By January 19, 1922 improvement had continued, so that most of the symptoms of which she had complained had disappeared. She had gained 16 pounds in weight and had remained sugar free without restriction in diet. Blood-pressure was 176/96. Further improvement and absence of glycosuria have been recently reported by the patient.

This is evidently a case of *non-toxic goiter of long standing which had recently become toxic; glycosuria and hypertension* were outstanding features. A subtotal thyroidectomy was done without difficulty under local anesthesia in this sixty-four-

year-old woman whose general health was poor. A marked decrease of the symptoms of hyperthyroidism and an apparent "cure" of the diabetes followed.

CASE VII (No. 95,813).—Mr. H. J. R., a farmer, thirty years of age, entered the hospital November 14, 1921. He had been neurotic since childhood, easily disturbed by trivial circumstances, and unusually sensitive. He feared that he might develop diabetes, of which there had been numerous cases in his family. He drank a great deal of water at times and passed much urine, which worried him. Shortly after he was married he became distressed concerning his family relationships, and was divorced. He spent a year of rest in California, but the nervous symptoms increased.

Loss of strength, nervousness, tremor, tachycardia, palpitation, sweating, and flushing of the skin were the chief symptoms. They came on indefinitely and gradually during the last three or four years.

Physical examination revealed an evidently neurotic individual without definite evidence of hyperthyroidism. The skin was hyperemic, face was flushed, and there was a marked tremor of the fingers. The thyroid gland was only slightly enlarged, uniform, without bruit. There was no indication of exophthalmos. The basal metabolic rate taken on two occasions was +54 and +47.

This was thought to be a borderline case difficult to differentiate from neurosis. Further observation and the increased rate of metabolism suggested the presence of definite hyperthyroidism. A subtotal thyroidectomy was performed by Dr. Kanavel on November 18, 1921. Under the microscope the specimen proved to be a colloid goiter.

On December 13th, almost a month after operation, the patient seemed definitely better. He had gained in weight and strength, was apparently less nervous, and had had no cardiac manifestations.

This illustrates a case on the borderline between a pure neurosis and hyperthyroidism. Many of the symptoms are common to both. To warrant a diagnosis of hyperthyroidism definite



physical findings are usually necessary. A high metabolic rate is a valuable and often the determining diagnostic sign.

CASE VIII (No. 93,913).—Mr. F. J. W., a locomotive engineer, fifty-one years of age, entered the hospital August 4, 1921. He had suffered for several years with attacks of vertigo ascribed to myocardial disease; he complained of mental depression, nervousness, irritability, and exhaustion on slight effort. For two months he had remained in bed on the advice of his physician. Digitalis, bromids, and valerian were prescribed, without benefit.

A small goiter had been present for years. Exophthalmos had never been noted. Loss of 40 pounds in weight; loss of strength; marked nervousness; generalized tremor; tachycardia; palpitation; marked constant sweating, profuse on exertion; flushing of the skin; irritability; frequent crying spells without apparent cause were complained of. All symptoms had been rather marked for four months, but the exact time of onset was indefinite.

The tonsils were large and evidently chronically infected, although he had not had tonsillitis for seven years. There was extensive infection about the teeth. The thyroid gland was small and uniform; no bruit present. The heart was enlarged, rapid (100 to 120); blood-pressure 140/70. The urine contained a small amount of albumin and occasional casts; Wassermann test negative; basal metabolic rate on two occasions was +60 and +57.

A diagnosis of *secondarily toxic goiter and focal infection (teeth, tonsils)* was made. The *cardiac manifestations predominated*. A subtotal thyroidectomy was performed August 9, 1921 by Dr. Richter. Sections showed an old colloid goiter with areas of hyperplasia and much round-cell infiltration. Convalescence has been slow. Two badly abscessed teeth were removed, aggravating the symptoms. Then an acute tonsillitis with manifestations of a general infection occurred; the patient was very ill. About one month later the tonsils were removed under local anesthesia without ill effect, although the patient was very nervous and drenching sweats occurred.



More recently 7 teeth were extracted. On February 10, 1922 the blood-pressure was 192/97. The postoperative metabolic rate determinations were: November 5th, +31; November 25th, +39; December 20th, +40.

March 10, 1922 the patient contracted the general respiratory infection epidemic at that time, and remained in bed for two weeks. On March 25th his condition seemed greatly improved; he seemed fairly normal, although still far from strong.

This case illustrates the clinical fact that patients with secondarily toxic goiter of long duration are apt to be considered cardiac rather than hyperthyroid. The cardiac manifestations predominate and it is because of them that the patient applies to the physician for relief. It also illustrates the close relationship between infections and hyperthyroidism.

CASE IX (Ambulatory).—Mr. F. H. L., an office worker, forty-eight years of age, was first examined in December, 1914 because of thyroid enlargement which had been present, stationary for one year, and then growing larger for two months.

A large thyroid gland without bruit; tremor; and tachycardia (120) were the chief findings. Potassium iodid, 8 grains three times daily, was administered for one month. At the end of that time the symptoms had disappeared and the thyroid gland had receded to its former size. He remained well until November 17, 1921, when he noticed a sudden and great enlargement of the thyroid gland, producing a sense of local pressure. He was nervous and irritable. Many teeth showed apical infection; the tonsils were small, but evidently chronically infected; the heart was rapid (rate 110); thyroid large, irregular, no bruit; blood-pressure 172/100; white blood-cells 14,900 basal metabolic rate +19.3.

A diagnosis of *secondarily toxic goiter*, probably adenomatous, and *focal infection of teeth and tonsils* was made. Thyroidectomy was advised, but refused. The patient consented to have the foci of infection treated. Seven teeth and later the tonsils were removed. Definite infection was found. Tincture of iodine, 12 drops three times a day, was administered.

By January 28, 1922, when the above measures had been

completed, the thyroid gland had receded in size. On March 2d his condition was definitely improved; the goiter was smaller; heart apparently normal (rate 80 to 90); blood-pressure 158/94.

While there is not sufficient evidence to warrant the assertion that the oral infection played a part in the production of hyperthyroidism, our experience has led us to believe that a definite relationship often exists and may well have been present in this case. Iodin, as a therapeutic agent, has proved valuable in this type of goiter.

CLINIC OF DRS. ISAAC A. ABT AND D. B. WITT

MICHAEL REESE HOSITAL

**A CASE OF CARBON MONOXID POISONING IN A CHILD**

**Carbon Monoxid Poisoning in a Child Five Years of Age; Total Blindness Remains as the Sequel. Carbon Monoxid Appeared in the Blood Fourteen Days After the Accident.**

I DESIRE to call your attention this morning to a little boy five years old who suffered a peculiar accident three days before admission to the hospital on February 7, 1922.

We are told that the little fellow was left alone in his home, and in some way, which is not fully explained, he succeeded in opening the gas jet and fell to the floor unconscious. He remained in this gas-filled room until he was discovered by the parents upon their return.

The nature of the accident was immediately discerned, and the boy was taken at once to a neighboring fire-engine house where one of the pulmotor machines was brought into action. Whether it was due to the pulmotor or not the boy was resuscitated in one and one-half hours. In the meantime, of course, the boy was in the open air, which fact contributed not a little to the resuscitation. The boy revived in a short time, became fully conscious, though he was weak.

On the day after the accident the little fellow commenced to vomit, and this continued more or less for several days. The mother also informs us that the child has grown progressively weaker. On the day previous to his admission he had several generalized convulsive seizures. His general condition became worse, and on February 10, 1922 he was brought to the hospital.

On admission it was noticed that he was stuporous. In his examination he offered no resistance and was completely relaxed. He appeared to be a well-developed and well-nourished boy. His cheeks were flushed; his lips and finger-tips were of a bluish hue; his respirations were slow and shallow. He was slightly feverish on admission and the thermometer showed that the temperature was  $101.4^{\circ}$  F. His pulse was of good quality, not accelerated, and 80 beats per minute were counted. His respirations were 20.

On examining the eyes superficially it was discovered that the conjunctivæ were injected; the pupils were small and equal, regular in outline, and reacted to light.

Upon examining the neck it was noticed that there was a slight rigidity of the muscles. The Brudzinski's sign was negative; there were no enlarged cervical glands.

The examination of the lungs showed that a few moist râles were present at the left base posteriorly, otherwise the examination was negative. The heart appeared to be normal in size; it was acting rapidly, but the tones were of good quality and the heart sounds were distinct and no murmurs could be heard.

The examination of the abdomen was normal.

Examination of the extremities showed that a moderate catatonia was present. The reflexes and knee-jerks were equal and brisk. There was a suggestion of a Babinski's sign on the right side, though it was negative on the left.

The urine was normal after repeated examinations.

The routine blood examination which was made during the coma showed no marked deviation from normal: the hemoglobin was 80; there were 10,800 leukocytes, and the differential count was practically normal.

You will recall the patient has been in the hospital for several weeks, and you will also remember that the little patient was comatose or semicomatose on admission. Several days after entrance into the hospital his condition became critical. The coma was markedly increased. The respirations were more rapid and shallow and the pulse more frequent. Cyanosis

became more pronounced and, indeed, his temperature rose from  $98^{\circ}$  to  $105.8^{\circ}$  F., and continued to rise until it reached  $107.4^{\circ}$  F. at 8.00 P. M. on the day that we are referring to. Numerous râles were heard over both lungs. His neck became rigid; his extremities were spastic; the abdomen was distended; his condition was threatening and the medical attendants expressed anxiety concerning his recovery.

At about this time the spinal fluid was examined and found to be clear, though the pressure was slightly increased. The globulins were negative and the cells were not in excess, and the majority of them were lymphocytes. The patient was given oxygen almost continuously by the use of the Benedict inhaler.

It was about this time that the little fellow showed violent twitchings of both eyelids; there was a tremor of the jaw, as well as of the fingers of the right hand. The twitchings also involved the muscles of the thorax and there was a spasmodic lifting of the diaphragm. The extremities became very rigid and the coma deepened. The child seemed very seriously ill. This was about the sixth day of his illness.

The eyes were examined by the attending ophthalmologist, Dr. Harry Gradle, who found external lagophthalmos, and also that the pupils reacted slightly to light. On examination of the fundi it was found that the veins were dilated and bluish red. Dr. Gradle remarked that there was a retinal cyanosis. About a week after this another ophthalmologic examination was made, and it was reported that the right fundus showed an optic neuritis; the vessels were engorged. In the left eye the outline of the disk was hazy. No evidence of optic atrophy was noticed.

The chemical examination of the blood at about this time showed that the carbon monoxid spectrum continued to be present. The carbonate combining power at this time was 63.5 c.c. per 100 c.c. blood plasma. This is said to be within normal range.

From this time on, about twelve or thirteen days after admission, he began to improve slightly. He answered questions intelligently, though he seemed unable to recognize objects;

his eyes were unaffected by light. It was also noticed at this time that he could not raise his right arm very well. The ophthalmologist who made frequent examinations reported that there was a high degree of secondary optic atrophy. The patient was unable to walk and he continued to show some weakness of the right hand.

In a few days he made a rapid recovery so far as the ability to walk and stand was concerned. He was feeling stronger and better in every way, though he continued unable to recognize light and the pupils did not react. He was completely blind. Arrangements were made to have him admitted to the state institution for the blind at Jacksonville.

The important points about the little fellow's history are that he showed marked nervous symptoms with coma; that the carbon monoxid hemoglobin continued for fourteen days after the accident, and that total blindness occurred, the result of an optic neuritis.

Clinical reports of children who have recovered from carbon monoxid poisoning are not of frequent occurrence in the literature. The pathologic changes produced by carbon monoxid are quite numerous and a variety of hypotheses have been offered in explanation. Various writers agree that the most common lesions found on autopsy are hemorrhages in the brain, degenerations, particularly of the lenticular nucleus and optic thalamus, thrombosis, and areas of softening in the central nervous tissue. Scattered small hemorrhages and intense hyperemia of all the body organs may occur.

McGurn has given a list of 105 varying pathologic conditions known to have been caused by carbon monoxid intoxication. A great variety of eye lesions has been reported including the following: central and retinal scotomata of the optic disks, color blindness, dipopias and engorgement of the retinal vessels, impairment of the pupillary light reflexes, toxic amblyopia, irregular pupils, nystagmus, edema of the optic disks, secondary optic nerve atrophy, retinal hemorrhages, and complete ophthalmoplegia.

Various views are held concerning the rôle played by carbon

monoxid in producing these lesions in acute cases. The older writers maintained that carbon monoxid exerted a specific toxic action upon the nervous system. More recent work, however, by Haldane, Henderson, and others tends to show that the continuance of coma, the subsequent tissue degeneration, and death resulting from carbon monoxid poisoning are not due to the retention of the gas, but are the results of injuries to the brain and other tissues by the deprivation of oxygen.

Henderson quite recently claimed that carbon monoxid is, in the strictest sense, not a tissue poison. He offers the following experiment as proof: Pieces of living and developing brain of a chick are suspended in a hanging-drop of chicken plasma at body temperature. Some of these hanging-drops are placed in an atmosphere of 79 per cent. carbon monoxid and 21 per cent. oxygen, while the controls are in atmospheric air which is 79 per cent. nitrogen and 21 per cent. oxygen. The former grow and develop quite as actively as the latter, although the carbon monoxid is 100 to 200 times as strong as would kill a chick by combining with its hemoglobin and thus asphyxiating it. The whole toxicity of carbon monoxid is said to depend on its combination with hemoglobin.

Applying these various hypotheses in explanation of the conditions which were presented in our patient, we may assume that the grave nerve symptoms which appeared about the sixth day were probably due to edema and hyperemia of the central nervous system, or possibly, as Edsall has pointed out, minute and scattered foci of hemorrhage, which gradually subsided without any permanent or residual effect upon the motor or sensory functions. The outstanding result in the little patient whom we have been considering this morning is the permanent blindness. You will recall that Dr. Gradle's examination showed an optic neuritis followed by atrophy. Whether this was primarily central it is difficult to determine. Very few cases of blindness are recorded in the literature. Other ocular lesions which have been reported include a case recorded by Sibelius in a boy of twenty who became permanently blind after carbon monoxid poisoning and who died three months

later from a degenerative and inflammatory process in his nervous system. The autopsy showed that the cortex was softened over a large area in both occipital lobes. Another case of total blindness is reported by Purtscher in a man sixty years of age. No changes were found in the fundi. Blindness was assumed to be due to small hemorrhages or areas of softening in the visual centers. There are also a number of cases recorded where temporary blindness occurred.

A case is reported of a boy six months old who suffered from carbon monoxid poisoning and who became permanently blind. The condition of the fundi is not reported.

Among other ocular complications may be enumerated homonymous hemianopsia, paralysis of the ocular muscles, xanthopsia, nystagmus, complete ophthalmoplegia, retinal hemorrhages, hemorrhages in sclera and conjunctiva, contraction of visual fields, partial color blindness, inequality of pupils, and irregular pupils.

In our little patient the carbon monoxid persisted for an unusually long time. It will be recalled that carbon monoxid hemoglobin was found as late as the fourteenth day, and the length of this persistence is unusual.

In this connection Henderson says that in the majority of cases practically all of the carbon monoxid is eliminated and the hemoglobin fully restored in three or four hours after the inhalation of the gas. Other reports, however, record the persistence of the carbon monoxid for a longer period; thus Fishbein reports having found carbon monoxid in the blood five days after the poisoning, and Wachholz reports carbon monoxid hemoglobin seven days after the inhalation.

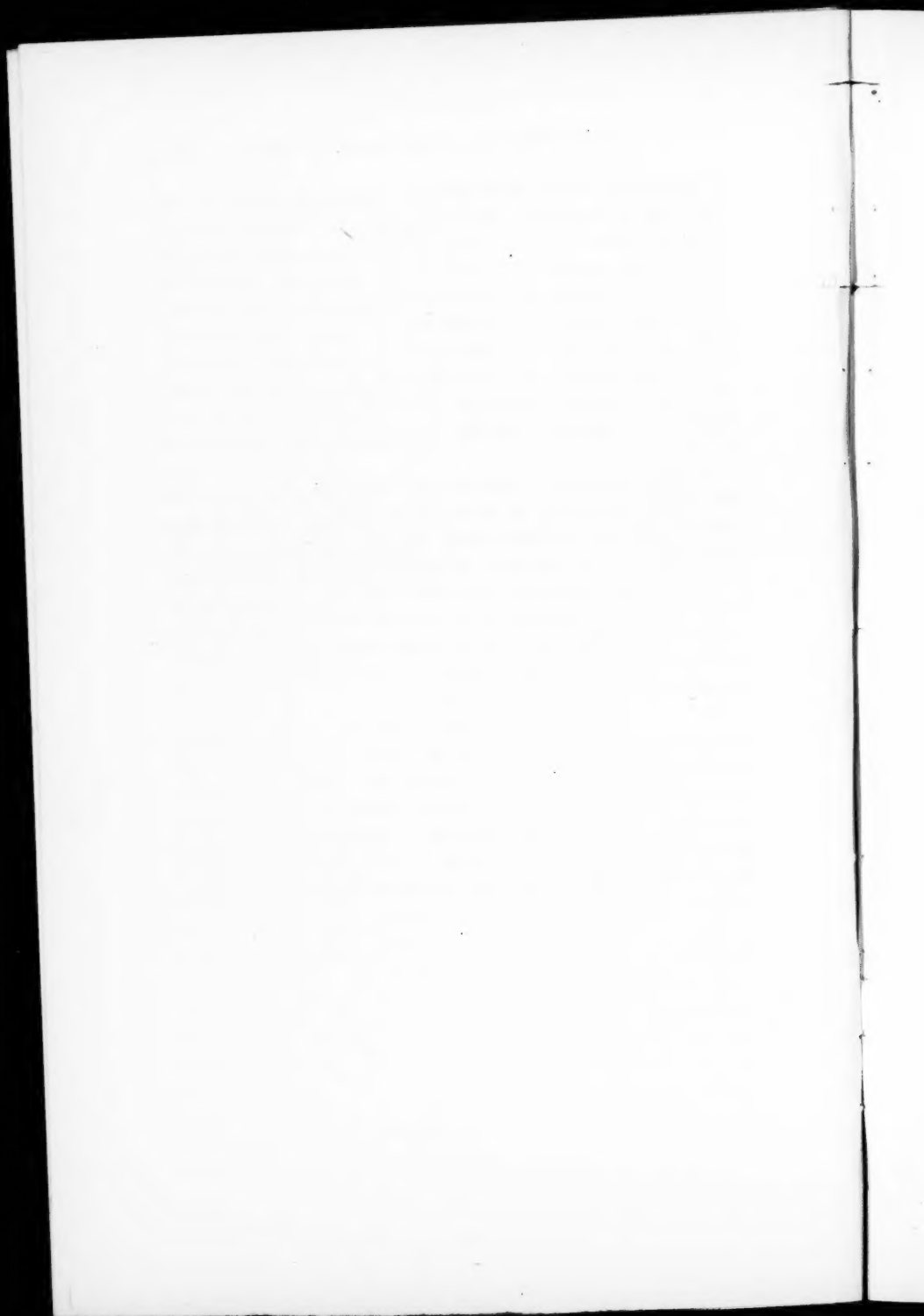
No very active treatment was pursued with our own patient except that oxygen was administered, as has already been stated. We considered the advisability of using blood transfusion as well as saline infusion. Blood transfusion was not employed in our little patient because at the time that it was under consideration his condition was so extremely critical and his temperature so high that we did not deem it prudent to attempt this form of therapy.



Henderson thinks that artificial respiration should be employed in the open air. He recommends the Schaefer method. He also advises the use of a new inhalation method devised by himself and consisting of 8 to 10 per cent. carbon dioxid in oxygen gas administered by a special apparatus. He believes that by this method the combination of carbon monoxid hemoglobin may be caused to disappear in ten or fifteen minutes. These experiments were performed on dogs. The use of the pulmotor as it was applied in our little patient is in no way superior to artificial respiration and, indeed, is not unattended by risk.

In this connection it may be stated that Henderson believes that blood transfusion or saline infusion are not rational procedures and are methods which are not applicable from the standpoint of his scientific investigations. In Henderson's opinion blood transfusion is not indicated, first, because he believes that the combination of hemoglobin with carbon monoxid is of very short duration, four or five hours, and second, because the red cells are not permanently or unfavorably affected by the inhalation of the poisonous gas.

We have shown you this little patient this morning because he illustrates the direful effects of carbon monoxid poisoning on the blood and the various tissues and organs, particularly the nervous structures. The damage which he sustained produced very severe, though temporary, symptoms for the most part. Permanent blindness seems to be a possibility in all of these cases, though, fortunately, it is not of frequent occurrence.



## CLINIC OF DR. JOSEPH C. FRIEDMAN

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### DIAGNOSIS OF THE GASTRIC NEUROSES

**Review of the Progress Made in the Diagnosis of Gastric Neuroses. Study of 38 Cases which Had Been Followed Over a Period of Three to Eight Years and in which the Diagnosis of Gastric Neurosis in the Strict Sense Had Been Made Originally After Careful Examination.**

THERE is perhaps no subdivision in medical nomenclature which has suffered more changes than that of neuroses, no matter what organ we may be considering. It is, of course, apparent that the general trend is in one direction, namely, a more and more sharp limitation of the symptom-complex included in that term. In other words, as our knowledge of normal and abnormal function broadens, the fewer are the classes of cases which have to be charitably clothed with this name to cover the nakedness of our ignorance. We need only to recall the fact that for Duchenne tabes was a functional neurosis, and a few decennia ago anterior poliomyelitis was in the same class. The first great advance in stomach pathology followed the introduction of the stomach-tube as a diagnostic agent. One might say that it brought forward more problems than it solved, as an immediate result of which the field of the neuroses seemed temporarily to be broadened. We had hyperacidity, achylia, continuous and intermittent hypersecretion, and, more numerous than any of these, those cases in which gastric symptoms were very pronounced, but in which the gastric secretion was often entirely normal.

The next advance was made by the development of abdominal surgery.

The third stride in progress was marked by the routine use of the x-ray in gastro-enterology, and it is the change in our conception of gastric neuroses brought about partly by that agent which I wish to discuss at this time.

According to Riegel, whose treatise is the classic of the pre-Roentgen period, gastric neuroses are defined as functional disorders that are not based on any pathologic changes of the stomach wall. He warns us that though we may be unable to discover any anatomic lesion of the stomach with the means at our command at present, still such negative evidence is insufficient. He adds that to consider the disease as a neurosis we should be able to determine that it is a disturbance of innervation, and predicts that many of the diseases then considered nervous will in time be found to be based on pathologic-anatomic changes, and adds that simply because a group of gastric symptoms does not fit into the frame of any of the anatomic forms so far discovered, we are not justified in styling it a neurosis or an anomaly due to perverted innervation. He, like almost all authors to date, follows Leube's classification of neurosis into secretory, motor, sensory, and mixed, the seat of the abnormal nerve irritability being either in the stomach, or resulting from disturbances in other organs, the so-called reflex neuroses of the stomach. Among the latter he includes those due to psychic irritation and emotional disturbance, as well as those due to organic diseases of the brain and cord, as gastric crises of tabes. The most frequent type of neurosis, according to Riegel, is the mixed, in which sensory, motor, and secretory changes are present, and its chief characteristics are, first, that the symptoms vary so frequently and in so many different ways, and second, that they are amenable to suggestive therapy.

Leube, besides his general classification, has given us a very definite conception of one group of those disturbances, viz., nervous dyspepsia. In this neurosis he would include all those disturbances which are limited to the *nervous apparatus* of the stomach. He says that the normal digestive act stimulates the central nervous system, causing a slight discomfort, feeling of pressure, etc. If these symptoms appear in unusual intensity,

and if others, as eructations, heartburn, headache, and dizziness are added, and gastric analysis shows normal motility but variable secretion, and the individual shows neuropathic tendencies, we may consider the disease as nervous dyspepsia. He adds that in such cases the central nervous system is in a state of great irritability, *i. e.*, reacts abnormally. His conception, then, is somewhat contradictory. He speaks of diseases of the nervous apparatus of the stomach, and then of abnormal irritability or reaction of the central nervous system. Which is primary and which secondary, or whether or not one may be present without the other, he does not tell us, and this, I think, is an extremely important point. Very striking in most articles is the absence of *any* attempt at a clear definition. Boas, for instance, heads his chapter "Nervous Gastric Disturbances," and evidently intends to include *all* anomalies of innervation due to local or general causes, and considers this vast group as synonymous with the term "gastric neuroses." He believes that the diagnosis is difficult, but that they are probably frequent, as the stomach is subject to so many disorders of innervation from its close connection with the large abdominal plexuses. One example will suffice to show the vast territory such a conception includes. Under the heading "Gastralgia Nervosa" he defines a disease characterized by intermittent attacks of pain in the epigastrium and independent of the intake of food. The pain is burning or boring in character, at times radiating to the hypochondria and back, sometimes severe enough to lead to collapse. Among the causes of gastralgia are: (a) those which arise from the stomach itself or surrounding organs, as ulcer, carcinoma, acid hypersecretion, pancreatic cysts, adhesions, etc.; (b) arising from central nervous system, as tabes, myelitis, and brain tumor; (c) infections and intoxications, malaria, nicotin, and gout; (d) reflexes from other organs: from the female genitalia, as retroflexion, ovarian tumors; and spermatorrhea in the male; ptosis of liver and kidney; (e) general neurasthenia and hysteria. In these cases one cannot find a definite cause. (f) Hernia of linea alba; (g) skin hyperesthesia; (h) gastralgia on an arteriosclerotic basis. We have given here

a list of practically all the conditions which may produce pain in the epigastrium or possibly in the stomach, and all such pains are considered under the neuroses. The principle underlying such a classification is difficult to determine and cannot lead to any clearness in thinking on this problem. Osler, too, has not defined the limits of the neuroses clearly enough. His chief criterion is a negative one, viz., the absence of discoverable anatomic basis for the disturbance in the stomach itself. He includes those cases dependent on a general neuropathic basis, but in his list includes apparently such divergent conditions as rumination, gastralgia due to menstrual disturbance, and the hyperacidity of chlorotic girls. It is difficult to understand why the hyperacidity of chlorotic girls is considered a neurosis of the stomach any more than the anemia is a neurosis of the blood-making organs. In the French literature one view commonly held is that expressed by Matthieu and illustrated by him in the case of gastric pains of nervous origin, viz., that they are due to hyperesthesia of the sympathetic ganglia in the abdomen. He believes that these ganglia form a reflex station and ordinarily prevent many of the nerve stimuli involved in the normal digestive acts from reaching the cerebrum. If the ganglia are in a state of exaggerated excitability and intensify the stimuli so that they reach the central station, then the individual feels normal acid, normal hunger sensation, normal distention, as painful. The concept is a clear one, but no explanation is given as to the cause of the ganglionic excitability. Glenard, of course, believed all functional disorders to be due to visceroptosis, with its mechanical consequences.

Dejerine gives an interesting classification into three groups: (1) Simple dyspepsia in neurasthenia in which normal functions are felt as distressful, and the individual becomes uneasy about digestion, has a repugnance for food, and by exaggerated attention magnifies the symptoms. (2) The class of phobias, in which the neurasthenic ends by concentrating his whole attention on his stomach, often with the aid of the physician. (3) The "Pseudogastropathies fonctionnelles," a class of cases definitely resembling organic disease, in which the symptoms have definite

relation to meals and are constant. The genesis of these complaints is at times from gastric disease originally organic, but prolonged, *e. g.*, women who have had disturbances during pregnancy.

This theory of the origin of gastric neuroses holds for the so-called nervous dyspepsia of Leube in which the gastric function is normal or nearly so. But suppose some constant disturbance of function is found, say hyperacidity, anacidity, or atony, how shall we interpret these? *A priori* there is no objection to widen our concept and consider these changes the result of abnormal *cerebral* stimuli. The very extensive nerve network in the stomach, indeed, might at first glance render such a theory probable. Certainly concepts with a strong emotional tinge can result in acute gastro-intestinal changes, as nausea, diarrhea, etc. There is also experimental evidence of their effect on secretion, as in Bickel's work on dogs with Pawlow fistulæ, in which during active digestion the sight of a cat (*i. e.*, the emotion it excited) caused a temporary complete cessation of secretion. Carnot, working with dogs with duodenal fistulæ, found that 200 c.c. of water normally passed through the pylorus in twenty minutes. At the sight of a whip there was immediate cessation of discharge through the fistula, evidently due to pylorospasm, while complete emptying took thirty minutes instead of twenty. Cabot believes that when a single symptom, such as eructation, diarrhea, or constipation, persists over a considerable period of time with little or no background of other connected symptoms it usually turns out that we are dealing with a neurosis, *i. e.*, a morbid habit.

It will be noted that in most of the previous definitions of neurosis the emphasis was laid on negative evidence, *viz.*, the absence of the findings of organic changes. There were, however, others who took a positive viewpoint, such as Beard and Strümpell. They believed that the gastric neuroses were simply a part of neurasthenia, and, therefore, while the demonstration of altered gastric secretion might be of little importance, the demonstration of altered psychology is quite essential. This, I believe, must be the basis of any clear conception of a neurosis.

We may define neurasthenia as a mental disease characterized by excessive susceptibility to fatigue, and emotional irritability of the central nervous system; also by physical symptoms due to abnormal emotional emphasis laid on various afferent stimuli. The neurasthenic need not be excessively easily fatigued, measured by objective standards. He need only *feel* so and be excessively distressed thereby. Then a gastric neurosis would consist of this same exhaustibility of the central nervous system, in which the neurasthenic or emotional emphasis is laid on normal or abnormal *gastric* function. In case normal stomach processes cause this abnormal psychical reaction we have a gastric neurosis pure and simple, *i. e.*, primary or psychogenic dyspepsia with all the criteria by which Leube judged his nervous dyspepsia, but in which all the emphasis is laid on the central nervous system. He notes, however, that pylorospasm is most easily produced by reflexes of intestinal origin, as from the duodenum. However, while temporary motor and secretory changes may result from psychical causes, it has never been demonstrated that long-standing ones are so caused. If, for instance, we have a hyperacidity or, more accurately, hypersecretion in a neurasthenic patient and no organic change is demonstrated, we are not justified in diagnosing a primary gastric neurosis, *i. e.*, a psychogenic disturbance of gastric function. Rather, we should think of the second and far more frequent class of cases, *viz.*, that in which *abnormal* stimuli from the periphery receive the neurasthenic emphasis in the cerebrum, in other words, a pathologic stomach in a neurasthenic individual.

Using this definition of neurosis as our guide, I think it will be worth while to go through the more important so-called gastric neuroses as ordinarily defined, to determine what justification there is for so classifying them and what is left after eliminating those not belonging here.

They are, of course, usually divided into the monosymptomatic and the polysymptomatic groups. It is evident that normal gastric stimuli may be emotionally heightened or lowered to result in a pathologic increase in appetite sensation or complete ab-



sence thereof, *i. e.*, bulimia and anorexia nervosa. Furthermore, that violent emotion may reflexly result in nausea and vomiting. Interesting in the latter connection are the cases seen during and after the war in army hospitals, in which the nausea and vomiting resulting from gas-poisoning kept up for abnormally long periods. Hurst regards those cases with a duration longer than two weeks as due to autosuggestion, and one can easily understand how the habit of vomiting is superinduced in individuals in such a state of mental and physical exhaustion. They are, of course, curable by psychotherapy. Nervous eructation is another justifiable diagnosis when a normal feeling of distention is centrally exaggerated and relief sought in the manner indicated, and when finally the memory of this sensation may be aroused by any unpleasant emotion, and the same reflex results. How often this happens we shall consider later.

**Nervous Gastralgia.**—I have already indicated to what extent the concept of neurosis is stretched in defining this group. We are not justified by any reasonable definition of neurosis in considering reflex pains originating in other viscera under that head, any more than we are justified in classifying a neuralgia of the fifth nerve due to a diseased tooth under the same head. It is a reflex pain or referred pain, and in neurasthenic individuals such pains may be felt more keenly or spread more widely, but they are not neuroses on that account. A very close analogy is found in the heart in what has been called vasomotor angina pectoris, in which attacks of angina pectoris occur in comparatively young individuals with normal hearts, from general vasoconstriction, say, from cold. They are not neuroses of the heart any more than the anginal pains occurring in the very slightly sclerosed heart of a neurasthenic is to be so considered.

Are there, perhaps, any characteristics by which so-called gastralgia could be recognized? Neuhoff gives the following: (1) They must originate or be located in the stomach. That is self-evident. (2) They must be neuralgic in character. Inasmuch as many, perhaps the majority, of neuralgic pains have a definitely organic basis, the neuralgic character is not of much

assistance. (3) The stomach wall must be anatomically free from lesion. That, of course, does not exclude the reflex pains. In short, if one wishes to have a very clear-cut, sharply defined class of stomach disturbances, in which there is no anatomic lesion in the stomach itself, but in which the pain, etc., may be due to disease of other viscera with which the gastric function is in intimate reflex relation, one is perfectly justified in so doing, and gastric pathology will gain by that, but one is not justified in including that group among the neuroses with which concept the idea of psychic origin is associated. In my own experience I have never seen a severe attack of pain, gastric in origin, which I felt sure was a neurosis. The following is an example of the close relationship between psychic trauma and organic disease. A woman aged forty, married, previously in good health, was greatly wrought up by the news of her daughter's elopement. A few hours later she began to complain of pain in the right upper abdomen; within forty-eight hours she developed jaundice; an operation a few days later revealed a gall-bladder with many stones. Here it was unquestionably the emotional disturbance which set in motion the motor impulses resulting in the impaction of the stone, causing the pain, jaundice, etc. One sees ulcer crises brought on by mental strain, but that, of course, is simply an exciting cause. I believe that discomfort is very often called pain by neurotics, and this difference between discomfort and severe pain is, I think, one of the distinguishing features of neuroses. I shall discuss this later.

**Hunger-pains and Pylorospasm.**—These are probably different names for the same disturbance, or perhaps different degrees of the same disturbance. It has been experimentally demonstrated that pylorospasm may be due to cerebral stimulation as in the case of the dog with the duodenal fistula, as above stated, but it has also been shown that it is far easier to induce pylorospasm by local reflexes as by a very minute ulceration in the stomach wall or irritation from the duodenal side. This is comprehensible if we remember that while the stomach is connected with the central nervous system by both sympathetic

and vagus branches, still it is capable of acting quite independently and the digestive function proceeds normally if its central nervous connections are entirely severed. It will be asked, however, how we can explain those cases of tumors of the pylorus described in the literature in which, at operation, either nothing was found, or a tumor of the pylorus was visible, which disappeared under the eyes of the surgeon, during anesthesia. The following will, I think, show the difficulties in such cases. A report was transmitted to the Congress of Internal Medicine in 1912 in which an x-ray diagnosis of scirrhus of the prepyloric region was made on a patient and the abdomen opened. A small tumor-like hardening of the lesser curvature was found with a chain of retroperitoneal glands. Resection was begun, evidently of the tumor and pylorus. When partly completed the tumor disappeared, and the resected pylorus showed nothing macroscopically after removal, but histologically the resected piece showed a small abscess which evidently was the cause of the pylorospasm. It is evident, then, that one must be very cautious in concluding that the stomach wall is anatomically normal. Probably many pylorospasms are to be explained in the same way, *i. e.*, by minute lesions of the mucosa or submucosa, which even careful search at a laparotomy may not reveal.

**Hyperacidity, hypersecretion, and achylia** are also considered at times to be neuroses. Many writers have, however, protested against this view. Hayem, for instance, states that in every case of hyperacidity on which he has held autopsies (death due to other causes, of course) he has always found glandular lesions microscopically. Moynihan states unequivocally that persistent excess of hydrochloric acid is an indication of organic disease rather than of functional. By the time one has eliminated the well-defined organic stomach disturbances and the reflex disturbances, including constipation, which may cause hyperacidity, certainly one finds few cases which could be considered a part of a general neurasthenia. As to hypersecretion, both continuous and intermittent, it is the consensus of opinion, I believe, that some organic change in the stomach

or central nervous system is invariably the cause of this condition in the more extreme grades. In the milder degrees we must bear in mind first of all the extreme difficulty of deciding what is normal. Carlson found that the rate of secretion of hydrochloric acid in the fasting normal stomach was from 2 to 50 c.c. per hour, and that a complete single aspiration of a fasting healthy stomach yielded from 0 to 150 c.c. of gastric juice. If we take the maximal figures here given, I am sure that very many mild grades of so-called hypersecretion will be covered. Such patients may still have gastric neuroses, but probably not many are of the so-called secretory type.

Ever since Einhorn described achylia gastrica, or complete absence of acid secretion, neurogenic forms of this condition have been described as distinguished from the cases due to atrophy of the gastric mucosa. The evidence of the nervous origin is of two kinds: first, the usual negative variety, *i. e.*, no gross lesion of the stomach could be discovered; and second, the fact that some cases have been described in which after a number of years of absent secretion hydrochloric acid has again been found in the stomach contents. Einhorn admits, however, that such cases are very rare, but mentions a more frequent class in which there is achylia one day and present hydrochloric acid another. In the latter instances there are two possible sources of error: one, that in a single aspiration only some saliva and gastric mucus may have been aspirated and not the test-meal; and second, as Curschmann has shown, if a more appetizing meal is served instead of the tea and toast, a certain number of so-called achylics will secrete hydrochloric acid. Again, the fear of the stomach-tube will at times inhibit secretion. Again, the change from the normal secretion to absence of hydrochloric acid happens at times during menstruation, to reappear again regularly in the intervals. Still more convincing are the histologic reports of Lubarsch. He found on examination of pieces of tissue aspirated from the stomachs of cases of anacidity invariably an atrophy of the specific gland cells with increase in interstitial tissue. Küttner found the same in histologic examination of postmortem specimens, and points out the

dangers of drawing conclusions from one small piece of mucosa, inasmuch as he found areas of normal mucosa and close to them areas of complete atrophy. With all this evidence, then, neurogenic achylia of any considerable duration must be an unusual occurrence. So much for the more important of the monosymptomatic group. We have left, then, the polysymptomatic group, or so-called nervous dyspepsia. I have already given Leube's definition of this term, and the picture described is, of course, a very common one. The frequency with which such a condition will be diagnosed as a neurosis will, of course, depend on the definition. If we include that large group of cases in which comparatively mild abnormal symptoms receive the neurasthenic emphasis, *i. e.*, an organic change of slight degree in a neurasthenic, of course, we may find many cases of nervous dyspepsia. The following is an example: A married woman, aged thirty years, complained of constant distress in the epigastrium, exaggerated by food, with some nausea occasionally, and some loss in weight. At home the pains were apparently so severe that she screamed aloud. In the hospital, while she complained bitterly of pain, close questioning reduced it to a burning sensation. She vomited a few times, but only small amounts of food. Gastro-intestinal examination, both by test-meals and x-ray, etc., was completely negative. Lungs and central nervous system revealed no organic changes. That we were dealing with a neurasthenic was unquestioned. Yet the disturbance was so persistent that a laparotomy was done, and after a very complete exploration the only pathologic finding was a slightly roughened, indurated right hepatic lobe, the gall-bladder being normal grossly and microscopically. The microscopic report on an excised section was chronic cholangitis, probably from a previous cholecystitis. This type of case is, I repeat, frequent. If, however, we limit the term "neurosis," as it should be limited, *viz.*, to normal function misinterpreted or emotionally overemphasized, these neuroses will, indeed, be infrequent. When one remembers that it has been demonstrated that disease anywhere in the body definitely affects gastric secretion and, as already mentioned, even physio-

logic changes such as menstruation and the menopause do the same, one will have to be very thorough in his examination to rule out some organic basis. The frequency with which neuroses occur has been variously estimated. Du Bois, Beard and Rockwell, and Dejerine, all, of course, more distinguished as neurologists than as gastro-enterologists, believed that 90 per cent. of the gastric disturbances are of psychoneurotic origin. Stiller believed that 25 per cent. were functional. I do not believe any writer of today would make such a claim, even including reflex gastric disturbances, and with the limits imposed by the definition given here, 5 per cent. would certainly fully cover the number of cases, I believe. That some of us err on the side of too frequent diagnosis of gastric neurosis the following statistics will demonstrate: A group of 38 cases was taken which had been followed over a period of three to eight years, and in which the diagnosis of gastric neurosis in the strict sense had been made originally, after careful examination. To justify the diagnosis there had been on the positive side always a marked neurasthenic state, and a history of marked psychic trauma; on the negative side, normal x-ray findings, absence of blood in the stool, and, of course, nothing in the original physical examination to account for the gastric disturbance. Those patients only are represented in this group whom I have followed with sufficient care for this period to warrant some decision.

Of these 38 supposed gastric neuroses, 27, or 71 per cent., have proved to have a definite organic basis for the gastric disturbance; 3 have remained in doubt, while 8, or 21 per cent., seem to be definitely gastric neuroses in the strict sense of the term. The largest group in these 27 cases was naturally formed by ulcer of the duodenum and pylorus, of which there were 7, as determined either by the history becoming more definite, x-ray positive, or by operation. Three cases were shown to be cholecystitis, 1 hepatitis of syphilitic origin, 1 hepatitis, probably from gall-bladder infection, 2 cases were chronic appendicitis, one, considered neurotic cardiospasm, was certainly a reflex, though the cause was not discovered. One case, diagnosed pylorospasm from x-ray evidence without defect, was

operated on, and the pylorus found to be normal. The symptoms still persist unabated. In that case it would have been wise to do a pyloroplasty or pylorotomy. I feel that that is the proper method of eliminating the probability of a minute lesion at the pylorus not found at the time of operation. One was a hernia, two physical exhaustion states, of the type Mackenzie calls the X disease. The remainder were scattered, including various conditions which may cause direct or reflex gastric disturbance, such as uterine fibroids adherent to the posterior pelvic wall, passive congestion (cardiac in origin), hyperthyroidism, etc. There was one case of achylia in a woman whose husband had syphilis, but who gave no signs of active disease and whose achylia did not improve under antiluetic treatment. She had a pronounced syphilophobia, was certainly a psychasthenic, but, of course, one is not justified in calling the achylia neurogenic on that account. The group of 7 (see p. 1666) which finally proved to be ulcers offers some interesting facts in comparison with the group of those which seem to be true neuroses.

It will be noted first of all that the pain or distress in every one of these cases is of the *late type, i. e.*, in contradistinction to the early type it came more than one hour after eating. Second, that it was more than simple distress or feeling of fulness; it was a real pain of moderate severity in all except Case II. Third, that night pain was present in 5 out of the 7 cases. Fourth, that motility was slightly impaired in 3 cases out of 7. By normal motility I mean the ability of the stomach to empty itself of an Ewald meal in two to two and a half hours, and of a pint of buttermilk with 5 ounces of barium sulphate in five to six hours. These 3 cases include all the impaired motility cases in the whole group of 37. In other words, where impaired motility can really be demonstrated, it is an indication of an organic disease involving the pylorus directly or reflexly, generally, of course, ulcer or carcinoma. The cases of impaired motility demonstrable fluoroscopically of the so-called functional or asthenic type are extremely rare. Fifth, that gastric secretion was normal in every case but one of this group. By this that the total



## DUODENAL AND PYLORIC ULCER CASES

No.	Age.	Sex.	Supposed etiology.	Characteristics of pain.				Gastric secretion.	Gastric motility.	Proof of diagnosis.
				Time relation.	Severity.	Night pain.				
2	24	F.	.	1½ hrs. p. c.	Not severe.	+		Normal.	Normal.	Operation.
5	59	M.	Mental strain.	Several hrs. p. c.	+	+		Normal.	Slightly impaired.	Course.
10	24	M.	.....	Late.	+	+		Hypersecretion.	Normal.	Course.
16	26	M.	Worry.	Late.	+	+		Normal.	Normal.	x-Ray, at first spastic duodenum relieved by atropin; later incisure at pylorus. Operation: Small pyloric scar and few fine adhesions.
23	55	F.	.....	Late.	Moderate.	0		Normal.	Impaired.	Course and later x-rays.
28	19	F.	.....	Late.	+	+		Normal.	Impaired.	Course.
36	33	M.	Worry.	Late.	+	0		Normal.	Normal.	Course.



acidity after an Ewald meal was at no time much over 60. In the entire 37 cases there were 8 cases of hyperacidity or hypersecretion. Of these, as stated, only 1 proved to be an ulcer. The rest were: 1 pulmonary tuberculosis, 1 cholecystitis, 1 hyperthyroidism, and 4 neuroses. In doubtful cases, therefore, the presence of hyperacidity does not help in deciding the presence or absence of an ulcer.

Case 16 is of considerable interest. It is one of two similar cases. A young man aged twenty-six was first seen in 1916, complaining of gastric distress, *i. e.*, feeling of fullness coming on about one hour after meals. There were no night pains and no nausea or vomiting. Secretion and motility were normal, x-ray findings showed no pathology. He was seen from time to time since then, relieved each time by dietetic measures. The last attack was more severe, the pains worse and night pains present; x-ray plates for the first time revealed a definite minute incisura in the prepyloric region of the lesser curvature. In view of the increasing severity of his symptoms a laparotomy was done. The operative findings were: a few old but fine adhesions on the anterior surface of the duodenum just beyond the pyloric ring, attached to a small puckered scar extending through to the mucosa. Microscopic sections of this area showed a typical healed ulcer covered by a single layer of epithelium. Here then was a healed ulcer producing progressively severe symptoms. These pains cannot be explained by the caustic action of acid on the injured mucosa, but only on the basis of reflex disturbances of peristalsis of the antrum and the pylorus resulting from this scar tissue.

The group of cases finally considered as neuroses included 8 (see p. 1668).

The age incidence is a little higher than that of the ulcer cases, averaging forty-five years as against thirty-four in the former group, and includes three times as many women as men.

The time relations of the pains is quite constant, only one case being noted as occurring one hour after eating, all the others coming almost immediately following the taking of food. The degree of pain is just as constant, in only one was it at all severe,

TABLE OF CASES FINALLY DIAGNOSED AS GASTRIC NEUROSES

No.	Age.	Sex.	Supposed etiology.	Characteristics of pain.				Gastric secretion.	Gastric motility.	Proof of diagnosis.
				Time relation.	Severity.	Night pain.				
4	50	F.	Mental stress.	Immediate p. c.	Not severe.	0		Hypersecretion.	Normal.	Normal for several years. Condition varies according to work and worry.
8	41	M.	Mental stress.	Early.	Moderate.	0		Hypersecretion.	Normal.	Perfectly well when not worrying.
12	33	F.	Mental stress.	Early.	Moderate.					
15	45	F.	Mental stress.	Late.	Moderate.	0		Normal.	Normal.	Perfectly well for several years except when under strain.
20	36	F.	Mental stress.	Early.	Moderate.	0		Normal.	Normal.	Course.
21	52	M.	Exhaustion.	Early.	+	0		Hypersecretion.	Normal.	x-Ray negative.
33	58	F.	Mental stress.	1 hr. p. c.	Moderate.	0		Normal.	Normal.	
26	46	F.	Mental stress.	Late.	Moderate.	0		Hypersecretion.	Normal.	Operation; nothing abnormal found; symptoms disappeared after removal of normal appendix.

in the rest being moderate, *i. e.*, a feeling of fullness or distress, never a severe pain; none complained of night pains.

A striking feature is the frequent occurrence of hypersecretion or hyperacidity, being present in 5 out of 8 cases, the acidity at times rising to 80 and over after an Ewald meal. The time of occurrence of this hyperacidity, however, was not constant, being present at times at the end of half an hour and at times only in an hour and a half, and is, therefore, of no value to us in distinguishing these cases. Motility was normal in every case. The frequent occurrence of hyperacidity in cases which seem certainly to be of neurotic origin is difficult to explain in view of the findings of Hayem of pathologic changes in such stomachs postmortem. It is probably not a coincidence, and if it has any meaning at all, indicates an excess of that part of gastric secretion due to nerve influence.

Taking for granted that the patient is demonstrably a neurasthenic, and that careful physical examination and minute x-ray examination has revealed no organic changes, except perhaps a hyperacidity, is there anything in the symptomatology of these cases which would enable us to distinguish the true neuroses from the reflex disorders and the organic gastric diseases? There are, of course, no pathognomonic signs. One fact is certain, and that is that none of the neuroses complained of a *severe* pain. They complained a great deal, of course, but one never obtained the impression of agonizing pain. Second, the symptoms observed over a period of time varied greatly in quality, the pain shifted its position, though the time relation to meals remained rather constant. Therapeutic effects were rapid and complete, though often temporary if the cause of the emotional instability remained. To repeat this statement from another viewpoint, those cases in which the pain really seemed severe, and the symptoms fixed in character and position, which remained uninfluenced by simple therapeutic measures, generally proved to have an organic basis.

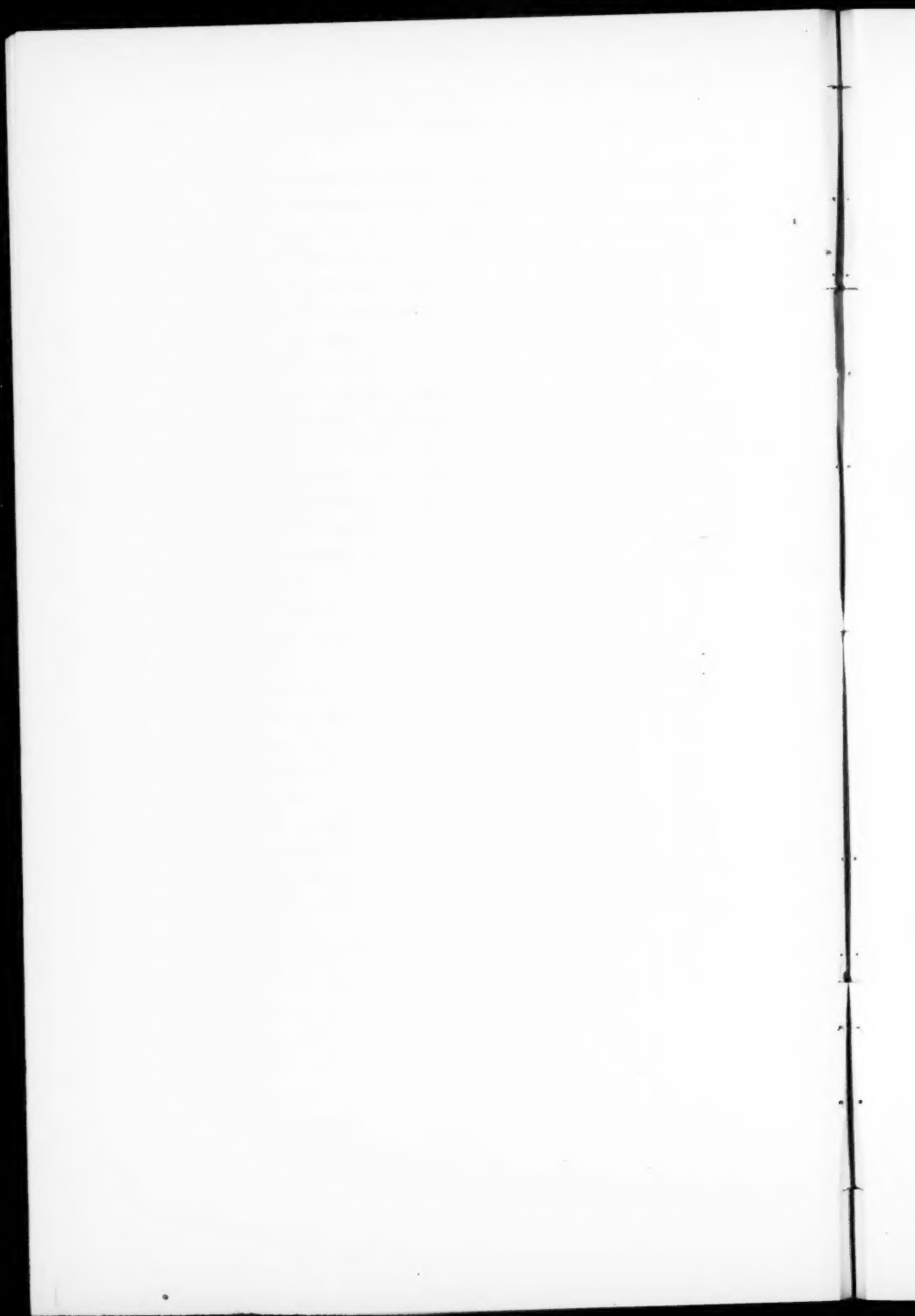
In considering the differential diagnosis of gastric neuroses I do not, of course, wish to go into detail; I shall mention only a few conditions which give those symptoms we call nervous

dyspepsia. One would say off-hand that slight lesions of the gall-bladder would be the most difficult class to distinguish from the neuroses, especially that not infrequent type of functional gall-bladder disturbance due to distortions of the cystic duct, and hence difficulty in emptying of the viscus. The points that I have mentioned as to severity and persistence of symptoms are apparently of value here, for only 3 out of the 38 proved to be cholecystitis.

Another group of cases consists of young people poorly built, poorly nourished, who are undergoing physical exertion beyond their powers. Some are clerks, others seamstresses, etc. Besides their backaches, which I believe some would term sacro-iliac disease, and lower abdominal pain, termed by others chronic appendicitis, and by others, again, due to focal infection, they have distress after eating (but never severe pains), with anorexia, nausea, etc. They improve as they gain in weight and physical endurance. They may or may not have viscerop-tosis in addition. I believe that the symptoms are due to muscle fatigue bodies, and, what is more important, that there is not necessarily a neurotic element involved. Indeed, it has been demonstrated experimentally by Manteli that after profound muscular fatigue the psychic secretion of the gastric juice is much diminished, while the direct secretion is minimal. Three hours of repose are necessary to restore normal secretion. They are closely related to the X disease of Mackenzie, only in those cases various vasomotor symptoms are added, such as acrocyanosis and functional heart disturbances. One such patient stopped work, felt quite well for three years, and then had a somewhat milder recurrence when she became pregnant. Another group very difficult or impossible perhaps to differentiate is that of ductless gland origin, especially in the menopause. The distress that comes after eating in many such cases is primarily due to absence of, or abnormalities, supposedly, of ovarian secretion, which, perhaps, through the sympathetic nervous system causes a relaxation of the intestinal musculature. This, of course, would no more be a neurosis than the deposit of subcutaneous fat resulting from the same cause.

There is, of course, in those predisposed a psychical change produced which may intensify the intestinal symptoms. The same argument holds, of course, for all gastric symptoms due to ductless gland disturbance, though we are less prone to think of the others as neurotic. Of chronic infections, especially the so-called focal infections, I shall not speak. There is at present no danger of this frequency being underestimated. On the contrary, they bid fair to occupy the position which the neuroses once did, and, to paraphrase Riegel, the tendency is to declare any group of symptoms that does not fit into the frame of the few disease forms so far discovered a focal infection.

To conclude, then, gastric neuroses, in the proper sense of the word, are rare diseases, almost as unusual, perhaps, as chronic gastritis, like which they have changed from an advanced to a very subordinate position in the scale of frequency of occurrence.



## CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

### ENDOTHELIOMA OF THE DURA

**Patient Giving History of Headaches of Six Months' Duration, With No Definite Etiology. Roentgenologic Examination. Physical Examination and Symptoms After Admission to Hospital Suggestive of Pott's Puffy Tumor of the Scalp. Operation—Findings. After-history.**

THE patient, J. M., thirty-seven years of age, was admitted to Mercy Hospital September 22, 1921. He had noticed that about six weeks previously he began having headaches. These headaches would ordinarily be present from the time he awakened in the morning until toward 11 or 12 o'clock, when they would disappear gradually, and in the evening he would be quite comfortable. The headaches for the most part were frontal, though at times he complained of localized pain in the left occipital region. The patient had no other symptoms. There was no vomiting, no disturbance of vision, and no complaint of disturbance of the central nervous system. His general health was good and his weight was 195 pounds; there had been no loss of weight. His sense of well-being aside from the headaches was perfect. It is true that he complained somewhat of a feeling that his head would stop up, but there was very little morning discharge from his sinuses. He had already been examined by a nasal specialist, who found the sinuses normal. His heart and circulation gave him no concern, though his pulse was a little fast, ranging from 86 to 94. He had no disturbance on the part of his kidneys and there was no nocturia. His alimentary canal gave him no trouble. His abdomen was

very comfortable and he was not troubled with constipation. He had consulted a physician in Philadelphia, who had concluded that the disturbance was occasioned by sinus infection because of the characteristic disappearance of the headache toward noontime, but treatment of the sinuses had given him



Fig. 298.—Roentgenogram, lateral view. Note the peculiar conformation of the bone in the left occipital region: A, Tumor.

no relief.  $x$ -Rays had been taken in Philadelphia and the sinuses were reported as being in fair condition.

After the patient had been in the hospital a day or two he stated that at times he thought his head swelled in a particular place. He had been of this opinion for some time because he had noticed that when he put on his hat it sometimes felt a little tight.



**Examination.**—On account of the complaint of headache, possibly due to sinus infection, we had an x-ray taken of the skull. The report from the radiologist was to the effect that the sinuses were normal. Curiously enough the slight disturbance in the bone which shows in the appended plates in the left occipital region (Figs. 298, 299) escaped the notice of the radiologist, because his attention had been directed entirely toward



Fig. 299.—Roentgenogram, postero-anterior view, of same case: *B*, Tumor.

the sinuses. On noticing this peculiar conformation of the bone in the left occipital region I thought necessary at once to have the fundi examined because of the possibility of a bony intracranial growth. This was done, and no choking of the disks, inversion of the color fields, or limitation of the field of vision was detected. The vision in each eye was perfect. Furthermore, there had been no vomiting which would suggest intracranial pressure, and his pulse-rate was so rapid as to make it

unlikely that there could be a brain tumor of any size present; else there would be bradycardia.

Examining the head, with special reference to the patient's complaint as to the swelling which he at times noted, it was found that there was a slight puffiness of the scalp in the left occipital region. Furthermore, on placing one's hand over this area and holding it there some time, it was evident not only to the examining physician but also to both the nurse and intern that the temperature was distinctly elevated. This at once suggested the almost forgotten malady, hardly mentioned in modern medical literature—*Pott's puffy tumor of the scalp*. As originally described this puffy tumor was really due to an edema of the scalp because of a purulent infection of the *diplöe* or *dura*. At once the question arose as to whether the patient might not have some such purulent process. His temperature, however, during the four days of observation before he was operated upon was normal, except for a temperature of 98.8° F. This was felt to be too slight to warrant any conclusion. The white count taken on entrance was 9000, and the differential count showed a normal distribution of the polymorphonuclear cells, small and large lymphocytes. The presence of headaches was easily accounted for and the occasional localization of pain which he had at first complained of posteriorly easily explained by the puffy tumor. It was accordingly felt that the only safe thing to do was to have this area operated on at once. If a collection of pus were present it should, of course, be immediately evacuated. If, on the other hand, it was found that the condition was a neoplasm, the earlier its removal the better for the patient. The urinalysis being normal and his general condition excellent as to heart and lungs, he was advised of the necessity of an operation and readily assented.

Operation was performed on September 25th by Dr. E. Wyllys Andrews. When the scalp was shaved the slight bulging in this area was much more evident than before the hair had been removed. When the incision was made around the area it was evident that the scalp was somewhat edematous. On peeling back the scalp to uncover it from the area of bone which

was clearly shown to be diseased by the x-ray, the scalp was found to adhere firmly to the outer table. It was, however, severed in the area of adhesion, which was about the size of a dime, and peeled back. The inner surface of the exposed scalp showed a peculiar appearance—a spot which was sharply demarcated, darker, redder, and drying more quickly when exposed to air than the surrounding healthy tissue. The outer table of bone was found to be roughened as though the periosteum had been denuded by some morbid process, and when the skull was trephined the instrument did not go from the outer table through spongy diploë into the inner table, but made its way through a peculiar kind of bone all the way from the outer to the inner table. After the trephine hole had been made and the edges enlarged it was found that the underlying dura presented the same appearance as the inner surface of the scalp. The pieces of bone which were removed were saved for further observation in the laboratory.

On enlarging the trephine opening to a diameter of about  $1\frac{1}{2}$  inches it was found that the dura was firmly adherent to the underlying brain tissue. It was impossible to strip the dura from the arachno-pia because dura, arachno-pia, and cortex were grown together in an indistinguishable mass. The edge of this apparent growth was so sharply demarcated that it was a comparatively simple matter to cut away dura and cortex to a depth which seemed to lie well below the level of cortical infiltration by the tumor mass. There was a good deal of hemorrhage of the lacerated brain tissue, but no serious difficulties were encountered, and in a few moments with pressure by sponges all hemorrhage ceased. Subsequently the diseased area of the scalp was also removed in its entirety, and this together with the dura and underlying brain cortex and pieces of bone were sent to the laboratory for examination.

All laboratory specimens showed the same picture. An endothelioma had apparently started from the dura and had grown solidly in both directions, toward the outside through the bone and into the scalp, and toward the inside, involving the brain. Sections of the scalp showed that all portions of

the endothelioma had been removed and so far as known all portions of the outgrowths into the skull were likewise removed, but unfortunately, as far as the brain is concerned, we found strands of endotheliomatous cells extending down along the blood-vessels as far as the section of brain removed. It was felt, therefore, that some of the endotheliomatous tissue was left behind, about some of the cortical vessels.

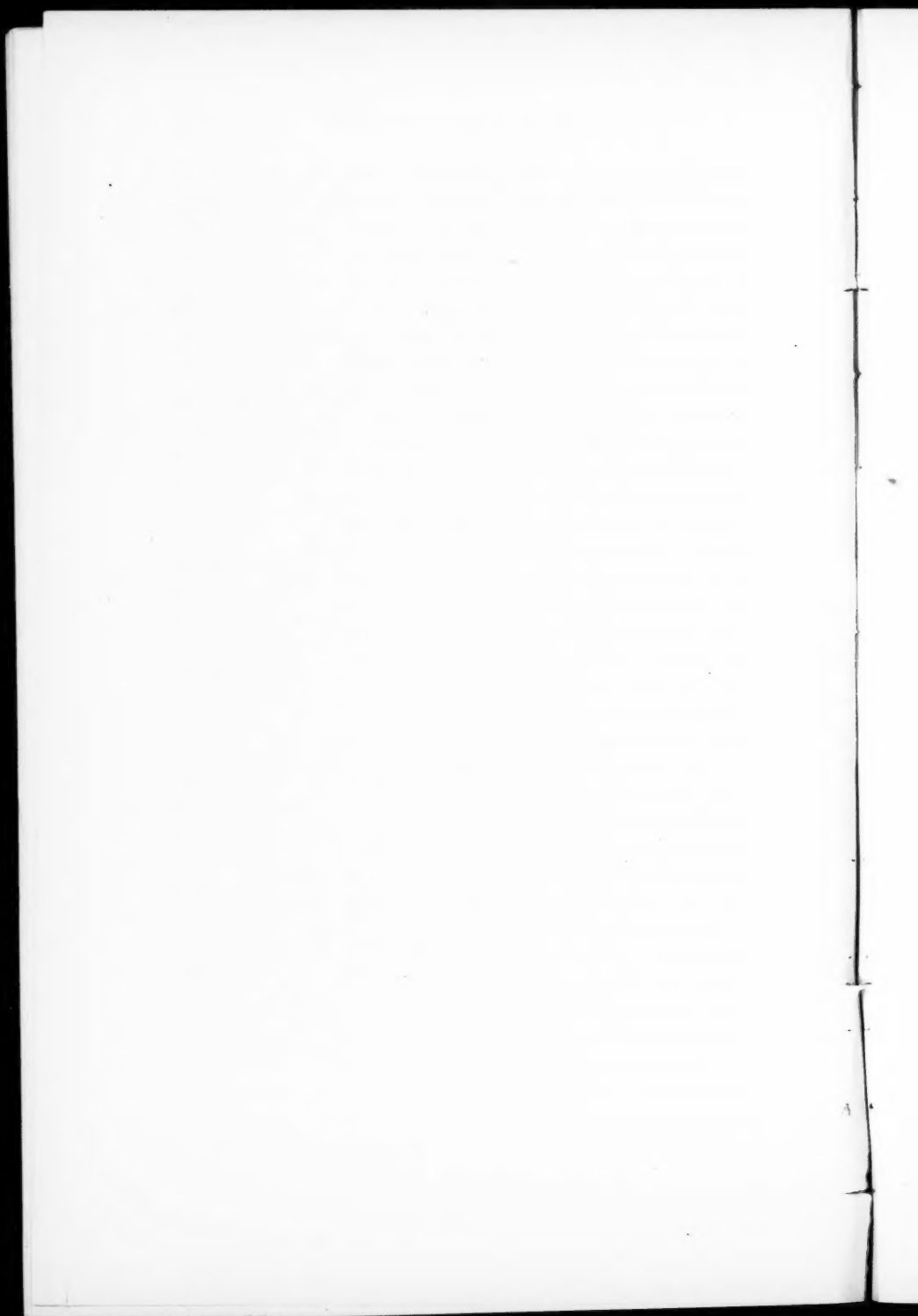
The patient's recovery was good. Though he never vomited before the operation, he did afterward, probably as a result of the etherization. The night following operation his temperature rose to 100.4° F. and his pulse to 118. He complained of much headache and he was quite restless, but he did not require any morphin until 2 A. M., when his restlessness became intolerable. The following day, the second after operation, the maximum temperature was 101° F. and maximum pulse 102. His kidneys and bowels both acted fairly well. There was some seepage from the operation, the bandage being slightly blood-stained, and the patient complained of headache, but on the evening of the second day he had several short naps and the nurse was able to record that the patient had a fairly comfortable day. The third day after operation his condition was quite good, although he complained of a good deal of headache now and then. He did not require any morphin and he never had more than two doses following his operation. Vomiting continued even to the third day, not because of any acidosis, for the urinalysis showed no acetone or diacetic acid, but evidently because of the disturbance to the brain tissue from the operation. After September 30th his condition kept improving with the exception of pain in his eyes. He began complaining of pain in his eyes on the night of September 28th and he complained of this pain continuously for several days.

Examination disclosed the fact that as a result of the operation and brain trauma he had acquired a homonymous hemianopia. He was unable to see the hand approaching from the right because rays of light were cut off from the left sides of his two retinae. This hemianopia has persisted to the present day and is the only disturbance which has resulted from the

removal of the brain tissue. It is likely that the trauma of the operation involved the higher visual cortex and it would suggest that the exterior occipital cortex is as much a center of ultimate sight as the cuneus on the mesial aspect of the occipital lobe. As the days went by he was permitted to get out of bed and to walk about the room, the headaches, on the whole, gradually diminishing until he was able to leave the hospital. Unfortunately the scalp did not heal very readily. Perhaps it had been too much clamped at one point at the time of the operation, and a portion of it sloughed away. The area, however, granulated in very rapidly, and at the present time the patient shows nothing but the scar of the operation and the pulsating area from which the bone has been removed. Though he occasionally has a little headache now he gives it no thought, and he has become accustomed to his hemianopia.

A very important postoperative treatment was the repeated use of heavy doses of the x-ray over the site of operation. As soon as the condition of the wound warranted these intensive treatments were started, and they were given to him at monthly intervals until January 1st, since which time he has been getting them at intervals of two months. It will be wise to continue these treatments in the hope of preventing any recurrence.

This case emphasizes the importance of little things. Before any choked disk had appeared, before there was any vomiting, before there were any eye symptoms of any sort at all, and while only headache was present, a diagnosis of brain tumor was made. Had the patient not been sent to the hospital, had the mere casual complaint of headache been passed off as it frequently is by physicians not too careful, this patient would have been lost to us and to himself. Had his complaint of a slight swelling, so that when he put on his hat it felt tight, been disregarded—and physicians are very prone to disregard symptoms as trivial—we would have missed the opportunity of recognizing a modern example of the almost forgotten Pott's puffy tumor of the scalp.



## CARCINOMA OF THE LUNG

**Carcinoma of the Lung in Patient Operated on Two Years Previously for Carcinoma of the Breast. Importance of Looking for Metastatic Conditions in Patients Giving a History of Previous Operation for Tumor. Treatment in Present Case.**

THE patient, Mrs. J. F. W., is fifty years of age. She is a very heavy woman, weighing 175 pounds. She comes to the hospital saying that she has had pneumonia and that she has been treated for it for six weeks, but that the diseased area is not resolving. She states that on January 29, 1922 in the early morning she was awakened with a dull pain in the right side toward the back in the region of the tenth and eleventh ribs. She never had such a pain before in her life. The pain gradually increased until at 2 o'clock in the afternoon it was at its height and it was very severe. There was no chill at the time and no cough attended the onset of the pain which lasted for several days. She thinks she had fever after the pain had been present for some time, but she is not absolutely sure of it because at the beginning until she consulted a physician her temperature had not been taken.

After she had endured the pain for some days a physician was summoned. He informed her on taking her temperature that it was 100° F. He furthermore stated on examining her that there was dulness in the right base and that she had pneumonia. He then treated her for pneumonia for about six or seven weeks. She has gradually and slowly improved up to March 21st, when for the first time she sat up.

On the occasion of her first getting up she developed pain in the left side of the chest at about the same level as the pain on the right side. This pain was very intense, lasting twenty-four hours. It was exactly the same kind of a pain that she had with the right-sided pneumonia except that it was more

severe. With the onset of this pain the fever reappeared and she became delirious. The delirium as described by her sister was of a queer sort, consisting more in taciturnity and negativism than in anything else. The sister states that the patient would not talk to her, possibly because she did not comprehend, but lay in a state of semicoma, and her friends in great alarm brought her to the Mercy Hospital Monday, March 26, 1922.

On entering the hospital she was at once examined. We found that she showed dulness in the bases of both lungs, but instead of presenting the usual bronchial breathing observed in the consolidation of pneumonia the breath sounds were, for the most part, absent in the bases of both lungs. Furthermore, in the lower portions of the more resonant parts râles were to be heard on both sides. Cross-examination showed that it was true that she had spat up bloody, rusty sputum from time to time, which had confirmed the attending physician in his diagnosis of pneumonia. But examination also showed that the whole left breast was gone. On making inquiry into her past history we found that she had the usual diseases of childhood with occasionally tonsillitis and inflammatory rheumatism, but aside from these early disturbances she had always been well until June, 1920, when her left breast was amputated. It was amputated because she had noticed even three years before that time a nodule within it. There had been no loss of weight. Her normal weight was 175 pounds and she had weighed this up to the time she was operated upon. Following the operation she got along very nicely and was given the usual postoperative x-ray therapy.

In January, 1922 another small nodule was discovered in the left axilla and was removed. From this also she made a good recovery, but in February came down with the pain in the right side of which mention has already been made. She declares that although she has had pneumonia she has not been specially short of breath. She admits that she has tired easily of late, preceding her illness, which she noticed the more because she has always been very strong all her life. She has been in the habit of getting up once each night for several



years, but there is no swelling of the feet or ankles or indications of failure to eliminate on the part of the kidneys. Her appetite has always been good and is good now. Her marital history has no bearing on her case and the same is true of her family history, the only point in the latter being that she lost one sister from tuberculosis and one from Bright's disease.

She brought with her on entering the hospital a urinalysis made on February 9th showing specific gravity of 1005, a faint trace of albumin, but no casts, red blood-cells, or other abnormal constituents. For some reason or other the total urea nitrogen, ammonia nitrogen, creatinin, and uric acid in the urine had been tested by the examining physician and found to be normal. The sputum had also been examined for tubercle bacilli, and a report dated February 24th says that none were found, although the smear showed a moderate number of leukocytes and a large number of bacteria, consisting of streptococci, pneumococci, and catarrhal micrococci. The blood examination made February 25th showed 60 per cent. hemoglobin, 3,570,000 red cells, 13,800 white cells, with polymorphonuclear cells 75 per cent., small mononuclears 16 per cent., large mononuclears 6 per cent., transitionals 2 per cent., eosinophils, 1 per cent.

The examining physician also had a blood-culture made and the laboratory report was to the effect that the blood-culture showed a pneumococcus. The blood-culture was made on February 25th and the positive result was noted March 1st. In this connection it might be well to say a word concerning laboratory findings. At the present time many physicians do not attempt to make a diagnosis. They merely appeal to a laboratory and beg the personnel to find something. Usually the laboratory technicians do find something, but they do not always find the right thing, and at times they make mistakes just as do clinicians. Why laboratories should always be looked upon as infallible in their statements and clinicians as very fallible is beyond my power to say. Certainly in this particular instance if pneumococci were found in the blood-culture they must have gotten there by some sort of contamination because this patient never had pneumococcic septicemia.

When one approaches a patient who has had a carcinoma of the breast with a recurring nodule which has been removed by a surgeon, attention should at once be directed to possible carcinomatous metastases. If you go into a field in which wheat has been sowed you expect to find wheat, and if you investigate a patient who has had a carcinoma and who is sick, you will expect to find carcinomatous metastases. In such a case one ought not to look for Bright's disease and make elaborate tests of urea nitrogen in the urine or immediately start making blood-cultures. One should rather first examine the patient, and then having found out what the patient shows on physical examination, investigative work should at once be directed toward these findings.

We heard the patient's statements of pain in both pleural cavities. We found dulness in both bases, as did the examining physician who took care of her, but we did not find evidence of consolidation because there was no bronchial breathing. We found rather a suppression of breathing, but the presence of râles indicated to us that the suppression was not due to fluid, but rather to some other cause. We therefore immediately asked that stereoscopic plates be made of the lungs. This was accordingly done, with the result that you will notice in the plate (Fig. 300), a beautiful round tumor mass in the left lower pleural cavity showing very plainly, and a larger, somewhat more irregular, and probably an earlier mass situated in the right pleural cavity. There is no escape from the conclusion that these masses are carcinomatous in origin. The slight temperature, the bloody sputum, the chronic character of the condition, the physical findings, and the absence of any evidence of disease elsewhere all preclude the possibility of the diagnosis being anything but carcinoma of the lung.

The main fact in all cases of this sort is the point of view. If the examining physician approaches his patient with mind wide open to all possibilities instead of being narrowed to one, he is much more apt to arrive at a proper diagnosis. Just because a patient shows dulness in the base of the lung, to conclude that there must be either a pneumonia or basal tuber-

culosis is wrong. All factors capable of producing such dullness should be examined into and, above all, if there is any doubt in the mind of the examining physician as to the proper interpretation of his findings, he should also have due regard to the x-ray plates and the fluoroscopic examination.



Fig. 300.—Roentgenogram showing carcinoma of left chest secondary to a carcinoma of the breast removed twenty-one months previously. Note the round tumor mass in the left lower pleural cavity, and the larger, somewhat more irregular and probably an earlier, mass situated in the right pleural cavity.

The patient preceding her entrance to the hospital showed a temperature varying from  $98.8^{\circ}$  to  $100^{\circ}$  F. and a pulse of 90 to 100. At one time the pulse-rate was as low as 86. At the time of her entrance into the hospital, where she arrived rather tired out, her temperature was  $98.8^{\circ}$  F. axilla, her respiration 32, and her pulse 128. She entered in a condition which

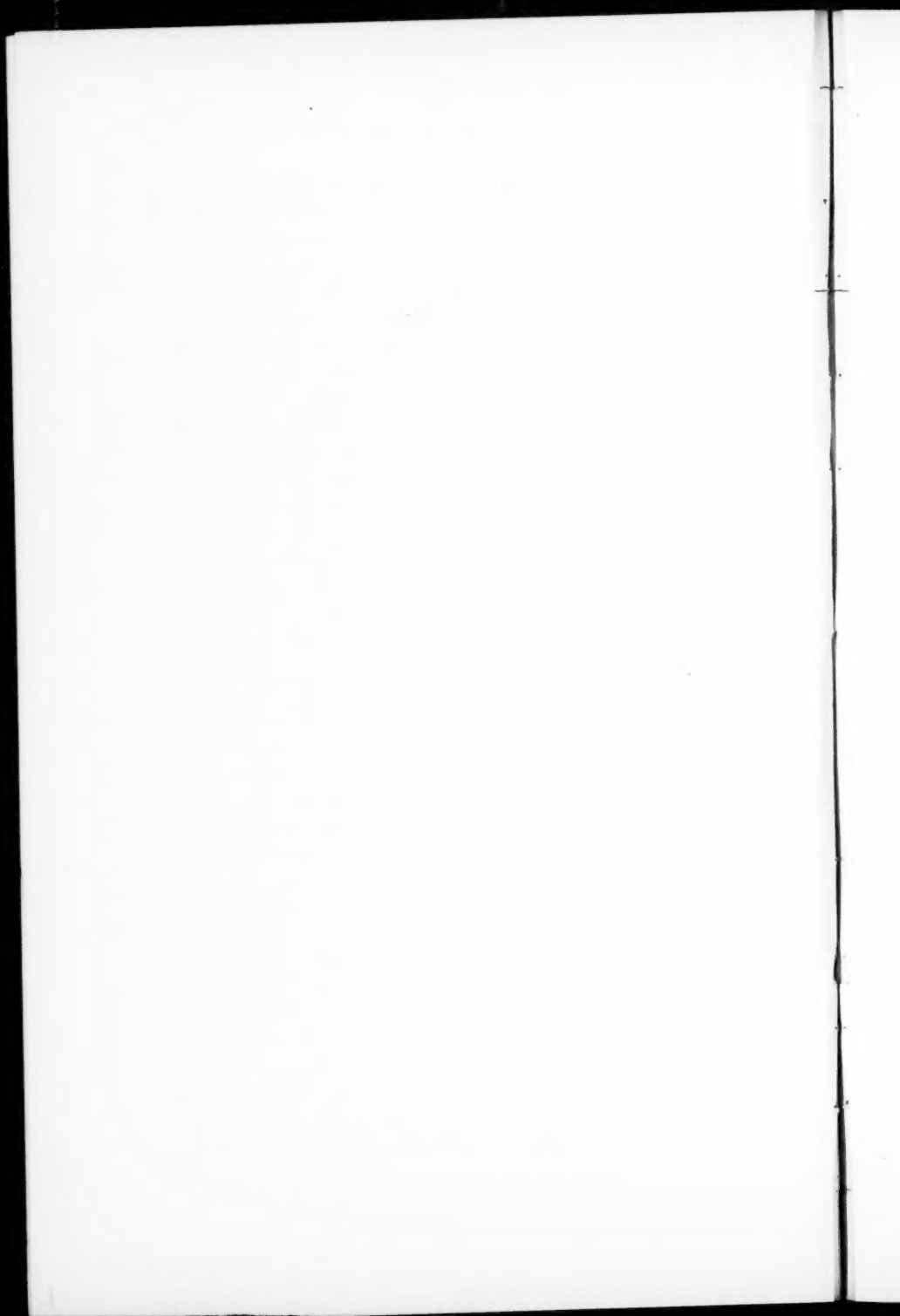
the nurse described as semiconscious, which is a much better term to apply to it than the term delirium applied by her sister. The nurse further remarks that she was irrational and that she had involuntary micturition during the night. The following morning her temperature was 99° F., her pulse 100, and her respiratory rate was 28. She was much stronger, but she was spitting up what the nurse describes as rusty mucus, which of course contained blood. She was coughing quite a good deal and had a good deal of pain in her left side. She slept only about two and one-half hours, but even after only this small sleep she seemed better and more rational. During the whole of March 20th she improved, her pulse being around 90 and temperature 97.8° F. During the latter part of the day she sweat a good deal, and this doubtless had much to do with lowering the temperature. The urinalysis on entrance showed a specific gravity of 1011, acid reaction, absence of albumin, casts, and blood-cells. A twenty-four-hour specimen sent to the laboratory the next day showed total amount 750 c.c., a specific gravity 1008, an acid reaction, a faint trace of albumin, no sugar, an occasional white cell, occasional red cell, and no casts. The respiratory rate on the 29th fell to 20 and the pulse to 82.

Very interesting was the behavior of the patient's temperature and pulse following the taking of the stereoscopic plates. This requires a little longer radiation than a simple picture of the chest because two plates have to be made. Before the picture was made the temperature was 99° F. and the pulse was 82. Three hours after the plates were made the temperature was 98.8° F. and the pulse 132. That night, however, the pulse fell to 102 and temperature to 97.6° F. and the respiratory rate to 22. The next day the patient declared she was very much better, and she ascribed, curiously enough, her improved condition to the x-ray, which was undoubtedly responsible. Her condition kept on improving from this rather transitory exposure, her cough became much less, the pain disappeared, and her sense of well-being returned, so that when I saw her April 1st, the last time before this case is described, she told

me that her only trouble was nervousness, and that her sister was wrong in being solicitous about her welfare, for she was all right except for her nerves, and that they would clear up and she knew she would soon be well.

Of course the patient has another fate in store for her. Nothing that physicians have yet been able to do has succeeded in curing metastases occurring in the lung. She will be given the benefit of intensive x-ray treatments and on Monday, April 3d, is to have a prolonged deep radium therapy lasting one hour. We feel, however, in advance that the results will be useless. The immediate results will probably be first rate, but the end-results will be merely a postponement of the inevitable. The patient needs no medication for the pneumonia because she has none. She needs no stimulation for her heart because it is doing very good work. Though she shows an occasional red blood-cell she shows no casts. We have not catheterized her because we do not regard it as scientific to ferret out in a patient with an incurable malady any possible microscopic metastases in her kidney by ureteral catheterization. It could readily be the case that the red blood-cells are due to a mild degree of passive hyperemia, notwithstanding the fact that the heart is fairly competent; at all events her kidneys and a possible red blood-cell in the urine are the least of her troubles. The main thing is the practical diagnosis and the practical treatment. As far as the former is concerned there is no question but that she has a carcinoma in the bases of both lungs and that the treatment should be by radiotherapy.

This case emphasizes the necessity of a prompt primary operation. This patient knew that she had a mass in her left breast three years before she had it removed. It was removed in June, 1920, now nearly two years ago, so that this patient has been living nearly five years with a carcinoma. Had the tumor been removed very early instead of in June, 1920 and had the operation been as radical early as it was when finally performed, the probabilities are that she would have been entirely cured of her trouble. There is only one cure at the present time for carcinoma, and that is its radical removal.



## ADHESIONS FOLLOWING CHOLECYSTECTOMY —CHIEFLY PERIDUODENAL

**Case I.**—Patient Giving History of Stomach Disturbance of Eight Years' Duration; Gall-bladder First Drained and Later Removed. Present Complaint More Suggestive of Gall-bladder Disturbance than of Stomach. No Improvement on Medical Management. Operation—Findings. After-history.

**Case II.**—Patient Presenting a Similar History of Gall-bladder Disturbance with Removal of Gall-bladder. Fluoroscopic Examination. Operation—Findings. After-history.

Discussion of After-treatment in Gastro-jejunostomy Operations.

THE above title is the diagnosis which was written upon the history sheet of this patient when she entered the hospital; and after our examinations were completed on December 28, 1921 we felt that the diagnosis was entirely justified by the history which the patient gave.

**Case I.**—The patient is a teacher, forty-five years of age. She had gone along in life as many people do, perfectly healthy until some eight years ago she began gradually to have trouble with her stomach. The disturbances which she had were ascribed chiefly to gas and belching, but she also complained of a good deal of irregular pain not related to the intake of food and not relieved by vomiting, except at times, and *not resulting in any loss of weight*. Strange to say, although her stomach bothered her from time to time, her weight even increased. She consulted Dr. J. B. Murphy at that time, and after examining her he concluded that she was suffering from a chronic cholecystitis and accordingly drained the gall-bladder. Her health improved and remained in better condition for some two years, when a recurrence led her to consult him again, and on this occasion he decided to remove the gall-bladder. This was done some six years ago, shortly before his death.

Again she experienced an improvement which lasted for several years, but in April, 1921 she once more began to have trouble with her abdomen. An area began to bulge through the muscles in the lower left side, and gradually there formed a large ventral hernia which was operated on by a surgeon who reported to me that the hernia was merely due to a weakness in the abdominal walls, that it had nothing to do with the scars of the first two operations, that it was in another part of the abdominal wall, that he had inspected the abdominal viscera and that they were in all respects in normal condition.

She recovered from this operation and went back to her duties, but was unable to carry them on after a time with any degree of earnestness because of more or less continuous disturbance with her digestion. She came to the hospital toward the end of December complaining of gas and belching which were very bothersome. There was no regularity in the periodic recurrences and the attacks themselves were usually of fairly short duration, suggesting a gall-bladder symptomatology rather than a duodenal ulcer. There was pain which was like the pain of a duodenal ulcer in respect that it came on about two hours or so after eating, but it differed from duodenal ulcer pain in the important respect that it was not relieved by the taking of food. In fact, the patient was unable to say whether food aggravated or helped it. To the best of her knowledge and belief it had not very much to do with it. The pain seemed to be a thing apart and of itself. Though at the beginning of her trouble the pain appeared two hours after meals, subsequently it would come on without any particular reference to meals, until at the time she consulted us she would have the pain anywhere from two to four or five hours after meals and even during the night.

The pain was not associated with undue constipation and in this respect resembled rather the pain of gall-bladder infection than the pain of duodenal ulcer. The most characteristic feature about it was the vomiting. I have often made the statement that cases of pure uncomplicated duodenal ulcer never vomit, and this I firmly believe. I have also long since



believed that if in a case of duodenal ulcer there is involvement of the peritoneum about the duodenum, so that there is a periduodenitis or pericholecystitis with traction adhesions between the duodenum and the gall-bladder, the patient as a rule vomits. However, in the case of this patient the vomiting was extreme. She usually vomited about two meals out of every three, so that she was continuously losing weight at the rate of about 2 pounds a week.

We accordingly made a thorough examination in the usual way, giving her a Rehfuß test-meal on one day and on the subsequent day making a fluoroscopic examination with plates. The result of the Rehfuß test-meal, specimens being removed at half-hour intervals for four hours, showed a hyperacidity both for total and combined acids, with a double apex to the curve. That is to say, the acid rose in the second and third half-hour periods, fell the fourth and fifth, rose in the sixth, reached its maximum in the seventh, and fell again in the eighth. This double peak is rather characteristic, in Rehfuß tests, of ulcer of the stomach or duodenum and of cases of hyperacidity in general.

Blood examination showed 70 per cent. hemoglobin, 4,380,000 reds and 8400 white cells. The differential count showed a reduction of polymorphonuclear cells to 54 per cent., an increase of the small lymphocytes to 34 per cent., 10 per cent. large mononuclears, 2 per cent. polymorphonuclear eosinophils. There was a slight acromia. Urinalysis showed a specific gravity of 1010, acid reaction, no albumin, no sugar, no casts, no red blood-cells, but an occasional white cell.

On making the fluoroscopic examination we found that there was no lagging of the barium in the esophagus, the stomach held normally and was fairly normal in position, reaching one handbreadth above the crest of the ilium. Peristalsis was very active. The antrum closed squarely to the tip, but the duodenal cap could not be seen and the emulsion passed through the first portion of the duodenum in a very thin stream. The duodenum was not freely movable and the patient complained of pain in this region when it was pressed upon. The

impression which we gathered was that the removal of the gall-bladder had brought about some fixation of the duodenum with the impeding of the normal duodenal flow. We observed regurgitation of the barium back through the second portion of the duodenum toward what should be the cap on several occasions. The radiograms taken immediately after the fluoroscopic examination showed some filling defects in the region of the duodenal cap and pyloric antrum, which we interpreted as being due not to carcinoma, but to distortion of this area by adhesions. The five-hour picture showed 25 per cent. of the emulsion still in the stomach, indicating that there was quite a degree of pyloric blockade. The twenty-four-hour radiogram, however, showed the stomach completely empty.

We accordingly came to the conclusion that the patient was suffering from adhesions of the second portion of the duodenum consequent upon the cholecystectomy some six years ago, and we were confirmed in this view by her subsequent history. She was very much opposed to operation, though we felt from the start that it was inevitable because of the pyloric blockade, and she was accordingly put upon an anti-acid diet. At first milk and cream were used, with no result whatever. For example, on January 2d she vomited 5 ounces after a breakfast which consisted purely of milk. She was then given malted milk, with the same result. For instance, on January 8th she vomited some of the malted milk. This showed a free hydrochloric acid of 24 and a total acidity of 45. Benzidin test for blood was reported positive. Several examinations of the feces, however, which were made from time to time during January, for instance, on the 11th and on the 14th and on other days, were all negative as to blood. I have before me the emesis sheets analyzed on January 16th and 18th, some of them showing a slight amount of acid, some a large amount. Repeated urinalyses were all fairly normal. On January 26th some blood was found in the feces, and again on January 28th, but this was interpreted by us as traumatic blood from straining while vomiting. In this connection I am reminded of a patient who was sent to this hospital because of gastric ulcer, who con-

stantly had for weeks blood in the stools. His physician feared carcinoma because of the constancy of blood in the feces. When I saw him it was evident that the blood came from bleeding gums. The man, being reassured that he did not have a carcinoma, gained in weight and became perfectly well. Frequently it happens that a positive benzidin test will be reported after examination of the feces, but laboratory reports must always be evaluated by the clinician. The blood is there, but the cause of the blood is not necessarily an ulcer. It may be one of many things.

The patient decided to persist in the dietetic treatment for quite a time and did so. We felt that her trouble was mechanical and not due to ulcer, and it seemed foolish to us to be rigid in regard to the food which she ate. We therefore took her off the milk diet and gave her a list of such things as she ought to be able to eat without exaggerating her natural tendency toward hyperacidity. It did not seem to make much difference with the different articles of food. She vomited with all of them and dropped in weight from 155 to 140 pounds in two months' time.

She finally returned to the hospital for operation, and was operated upon on March 20th by Dr. E. W. Andrews. A number of adhesions were found making it very difficult to get at the pylorus. The pylorus was, however, ultimately freed of those adhesions which had bound it down most firmly and which had mechanically interfered with its lumen. Though the adhesions were in part removed it was impossible without doing too much violence to the abdominal viscera to free them completely. It was, however, possible to do what we had originally planned, namely, a gastrojejunostomy. With adhesions about the pylorus which so matted it down that the pylorus was about 25 per cent. incompetent it was really foolish to attempt to release them, because within a week they would all return with interest. The wise thing to do was to release the adhesions just sufficiently to do the necessary gastrojejunostomy. This was done and the patient's subsequent history will be mentioned later.

**Case II.**—The second patient was almost the counterpart of the one just mentioned except that she was a young woman of twenty-six years of age. This patient was referred to us for examination early in December. The physician who brought her to us was at his wits' end because of her gastric distress. She was originally operated upon about two years ago for cholecystitis. The physician declares that she had an abscess in the gall-bladder area from some infection, that this abscess was drained, and the gall-bladder was subsequently removed. We have never been able to get a perfectly accurate history of exactly what happened. The process was evidently an acute suppurative one, but how a young girl, then twenty-four years of age, could get such an infection out of a clear sky without previous disturbance of health, as was the case with her, is difficult to discover. At any rate following the operation of cholecystectomy she entered upon a life of invalidism. Her complaints were very numerous and very puzzling. They might be divided into two general groups, those on the part of the nervous system and those on the part of the alimentary canal.

The nervous complaints were frankly hysteric, so much so that one neurologist who saw her was of the firm conviction that all of her trouble was in her head. She would spend a large part of her time in bed complaining of abdominal pain and wringing her hands. She would cry a good deal and was in a terribly depressed state. She made life miserable for her attending physician by calling upon him at all hours of the day and night. He would often get up at 4 o'clock in the morning and go over to her house to pacify her. He was more than anxious that something radical be done for her, both for her sake and for his own peace of mind. He was sure there was some organic trouble, but he was uncertain as to what its nature might be.

On one occasion for a period of a week she complained of a great deal of headache and vertigo. She would arch her back in a mild degree of opisthotonos and vomit incessantly. At the same time she ran a slight amount of fever, the trunk muscles

were quite rigid, and she was extremely sensitive to touch. With the presence of headache, fever, vomiting, hypertonicity, and hyperesthesia he had almost come to the conclusion that she was suffering from meningitis, and I was called into consultation. On examining her there was no evidence of real meningitis; indeed, there was not enough evidence to do a spinal puncture, which, of course, one would hesitate to do in such a patient because of the great increase in the nervous manifestations which it would undoubtedly call forth. Her temperature subsided after a day or two, and she got into a somewhat improved state of health, but still complained very much both of nerves and of stomach. Her gastric complaints were chiefly those of gas, belching, burning, pain, and vomiting. There was no periodicity about the attacks. For instance, in the beginning the seizures lasted about three or four weeks at a time, appearing at intervals of two or even six months, but in the later stages of her trouble the attacks of pain were of most varied occurrence. The weight fell from 140 to 106 pounds.

Besides the pain, gas, and belching there was intolerable vomiting, vomiting which so shortly followed eating that she refused to eat, and starved herself to such an extent that she actually when we examined her had a starvation acidosis with both acetone and diacetic acid present in the urine. She was extremely tender in the duodenal area. She was brought to the hospital and given the usual laboratory and x-ray examinations. The Rehfuess test-meal showed a high degree of hyperacidity with a double peak curve. Blood was not present, neither was bile in the stomach contents. Examination of the feces was negative as to blood.

The fluoroscopic examination was extremely interesting. It showed a low-lying stomach with a very much distorted duodenal cap which would not fill properly, which was tender on manipulation and which was immovable. The most interesting thing, however, is disclosed by the plate (Fig. 301), which shows a very sharp angulation between the first and second pieces of the duodenum. But one plate appears in this account, although several were taken at various intervals of time, and

all of them showed the same angulation of the duodenum. Furthermore, on watching the duodenum we could see a regurgitation of the stream back toward the cap from time to time. It appeared that the duodenum was narrowed at its



Fig. 301.—Roentgenogram (Case II) showing a very sharp angulation between the first and second pieces of the duodenum.

second portion to a diameter not much greater than that of a lead pencil, with the result that the stomach delayed its emptying, emptying rather slowly and with considerable difficulty.

In reviewing the history it is evident that the gas and belching were evidences of hyperacidity, that the type of periodicity is no evidence of ulcer of duodenum, stomach, or gall-bladder infection, and besides the gall-bladder had been removed. Vomiting is not a symptom of pure duodenal ulcer, but is a symptom of peritonitis or traction upon the peritoneum about the duodenum and gall-bladder in the upper right abdominal quadrant. Whenever a peritonic process interferes with the rich peripyloric nerve supply such reflex vomiting is very apt to occur. We interpreted the repeated vomiting in this case as partly due to acidosis and partly due to periduodenal adhesions. The patient was extremely constipated, this being indicative either of ulcer of the duodenum or periduodenal adhesions, constipation being absent in the ordinary case of cholecystitis and cholelithiasis. The loss of weight indicated an organic malady rather than a functional one, notwithstanding the abundance of the functional disturbances. The absence of blood in the feces and in the stomach contents indicates an absence of any ulcerative process and rather strengthens one's idea of a tied-up duodenum. The conclusion, therefore, was reached that she was suffering from periduodenal adhesions secondary to cholecystectomy and operation was advised. Although she had had a stormy time from the previous operation, she finally consented, owing to the miserable state in which she found herself.

She was operated upon March 21st. At the time of the operation by Dr. E. W. Andrews the adhesions found were even more marked than in the case of the preceding patient. It was impossible to free them; indeed, it was difficult even to release them sufficiently to make possible a posterior no-loop gastrojejunostomy, the operation that had been decided upon in advance. It was found on inspection that this was the operation indicated and it was accordingly performed.

#### POSTOPERATIVE TREATMENT OF GASTROJEJUNOSTOMY CASES

We have learned in the last few years a good deal in regard to the postoperative treatment in gastrojejunostomy cases,



so that now we have almost a routine. We do not believe in routine because every case is a case unto itself, but by routine we mean the general rule of procedure which can be followed in almost every case, but which may be varied depending upon the individual needs of a patient. A few years ago at this hospital a case of gastrojejunostomy was occasionally lost on the fifth day by what was diagnosed as pernicious vomiting. These cases were fatal, not from any oozing of blood, as we once thought to be the case, or from any failure in the technicalities of the operation, but was really physiologic, due to the then unknown advent of acidosis. By acidosis we do not really mean that the patient's blood becomes acid, but we mean that the blood is rendered less alkaline than it ought to be. The classic signs of acidosis in the text-books always include dyspnea, but long before the stage of dyspnea is reached there is more or less pronounced vomiting.

If at the time of vomiting the urine is examined it will almost invariably be found to contain acetone and diacetic acid in much greater than normal amounts. There are three reasons for this state of affairs. Almost all individuals requiring gastrojejunostomy have been more or less starved before operation either by voluntarily or involuntarily withholding their food. The result is a mild degree of starvation acidosis, and if the examining physician tests the urine of these patients he will find acetone and diacetic acid even before operation. Following operation two other causes for acidosis turn up. One is etherization, which will frequently cause acetone and diacetic acid to appear in the urine, but more important is the withholding of food, which is characteristic of the after-treatment of all cases. The absolute starvation which follows gastrojejunostomy leads to very many acidosis cases, so that because of the three causes—starvation preceding operation, etherization, and starvation following operation—practically every one of these patients shows acetone and diacetic acid in the urine.

Virtually every one of these patients vomits. They vomit following operation first as a result of the ether, and second



as a result of the oncoming acidosis, and this vomiting unless checked becomes more and more marked, even pernicious, yet it is extremely easy to check. All that is required is not gastric lavage, which is traumatic and more or less unsatisfactory, but the loading up of the blood with bicarbonate of soda and glucose. It is our custom now to begin before operation with the deacidizing treatment. We give to these individuals preceding operation glucose and bicarbonate of soda. If they can take it by mouth they get it in that manner; if not they get it by proctoclysis. Immediately following operation the treatment is continued in this manner: Four, 500 c.c. injections of 5 per cent. glucose and  $\frac{1}{2}$  per cent. bicarbonate of soda are given in each twenty-four-hour period by proctoclysis, at a six-hour interval. I find that many nurses and even physicians are quite hazy as to the rapidity with which proctoclysis should be given. I found a nurse in another hospital not long since given proctoclysis 10 drops to the minute. Ten drops to the minute means 1 dram in six minutes or 8 drams or 1 ounce in forty-eight minutes. It would take over six hours to give 8 ounces,  $\frac{1}{2}$  pint. It is absurd to give proctoclysis in this manner and then to have the nurse tell the physician that the patient retained only 300 c.c. They have done well to give even 300 c.c. The drip should be at the rate of 45 drops per minute. This gives almost an ounce every ten minutes or in the course of an hour nearly 6 ounces and in two hours about 12 ounces. In this way the patient can take easily within three hours' time 500 c.c. and have three hours' rest. Occasionally it happens that the rate will have to be reduced from 45 drops to 40, 35, or even 30, but it ought not ordinarily ever to be less than 30 drops per minute.

No special care is required in the preparation of the glucose. Strained honey may be used and it is perhaps the best glucose, but if this is not available Karo syrup is all right. Personally, I prefer honey because it is a mixture of natural glucoses and they are absorbed into the blood-stream without any digestion whatever, because they are pure dextrose. Bicarbonate of soda is also thoroughly absorbed, and this adds base to the

body which can subsequently unite with the inorganic acids and be eliminated, thus reducing the acid. It is our custom to give 2000 c.c. by proctoclysis in this manner for three full days. If the patient on the third day is quite free from signs of gastric distress we may permit sips of warm water. We always permit the mouth to be rinsed as freely as the patient wishes on the first, second, and third days. We caution the patient not to swallow the water, but we occasionally believe that the patient does so. We do not, however, think that very much harm is thereby occasioned, providing warm water is used. Cold water apparently brings on peristaltic contraction and pains. Warm water does not seem to have such an effect. We are beginning to believe that water can be given freely on the third day in many cases, and possibly we shall as we become bolder permit the patients to have a certain amount of water on the second day. On the fourth day we begin mouth feeding. We give them by mouth the difference between the amount they take by proctoclysis and the 2000 c.c. which we regard as necessary for existence; on the fourth day at least 500 c.c. are given by mouth and 1500 c.c. given by proctoclysis. We give on the third day apple cider, honey and water, orange juice, and grape juice. We choose these things because they all contain dextroses. If cane-sugar is used it has to be digested, and patients with acidosis as a rule are incapable of digestion. If, however, dextrose mixtures are used, the dextrose as such is absorbed into the blood-stream where it exerts its beneficial action. On the fourth day, providing we have given the patient some dextrose on the third day, we may permit the patient to have clear broths. On the fifth day our patients are permitted to have clear broths, thin gruels, plenty of fruit juices, plenty of water, and tea if they wish it. On the sixth day we permit creamed toast thoroughly soaked so that it is perfectly softened, crackers which are thoroughly soaked in hot milk, various broths, gruels, fruit juices, and tea.

Beginning with the operation every single specimen of urine which is passed is sent to the laboratory for individual determination of acetone and diacetic acid. The physician

who follows up each specimen of urine will find that the acetone and diacetic acid disappear from the urine when the patient ceases to vomit, and with any return of the acetone and diacetic acid there is a return of vomiting. Indeed, vomiting runs so parallel with acetone and diacetic acid that one can predict from the urinalysis whether the patient is vomiting or not; or, if vomiting, whether acetone and diacetic acid will show in the laboratory report. After the third day acetone usually begins to disappear. It is frequently absent on the fourth day and thereafter. Occasionally after being absent in one or two specimens it will reappear, but ordinarily only for a short time.

Both of the cases which have been demonstrated have been on this plan of treatment, and both of them have had wonderfully easy convalescences. In the case of the teacher there have been but two vomiting spells since the operation, and I think no more than that in the case of the young woman. This contrasts very markedly with our earlier experience and with the memories which we have of interns rushing for stomach-tubes and washing out the stomach in these cases. Gastric lavage is no longer necessary, because by controlling the acidosis there is no vomiting.

In this connection it may be perhaps worth while to cite how I happened to start using apple cider in the cases of acidosis. So far as I know I am the first one who has made use of it, though now it is being used in a great many places in post-operative gastrojejunostomies. The pure apple cider should be used, not the ordinary synthetic sort which one occasionally encounters. Cider out of a barrel from a grocery store is very apt to be a better sort of cider than some of the more expensive bottled ciders, though even barrel cider is occasionally not a strictly pure product.

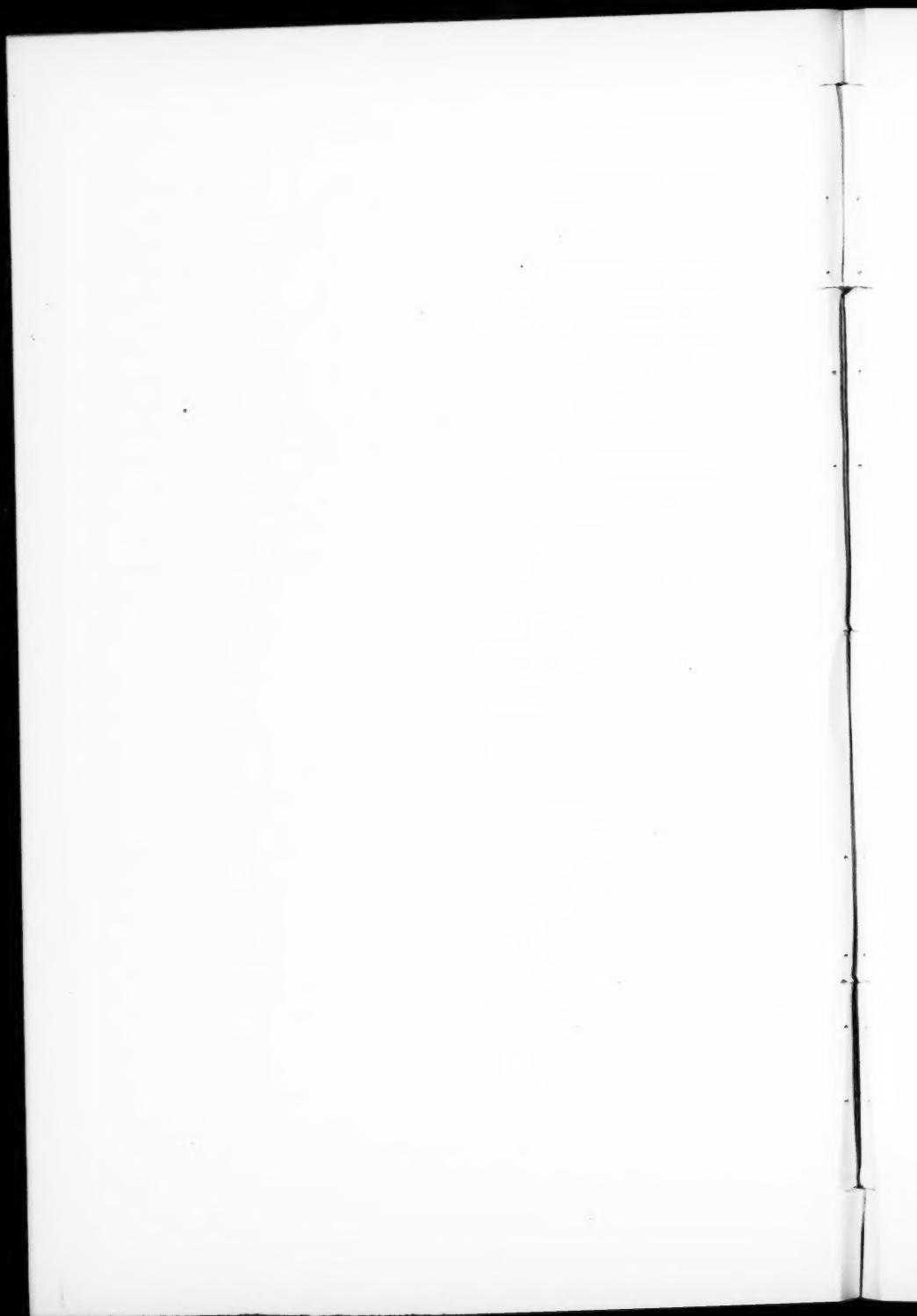
Some years ago when I was a medical student there was a patient in the Massachusetts General Hospital, twelve years of age, who was said to be suffering from hysteric anorexia. She was the daughter of a Bostonian of ample means. Her attending physicians were my teachers, Drs. Porter and Reginald H. Fitz. Everything that money and science could devise

were used to save her life, but she was slowly sinking. The attending physicians were in no doubt as to the correctness of the diagnosis of hysteric anorexia, and daily saw their patient slipping from them. Anything she took by mouth she immediately vomited and even water came up. Finally, on one occasion she rather weakly observed to them that she believed she could keep down some cider. This was just a slight instinctive desire on her part. Since all else had failed, the cider was forthcoming. She drank some and retained it. She drank more and kept that down. Finally in two or three days the cider had her stomach so far under control that she ceased vomiting and ultimately recovered. With a smile our professors told us that the diagnosis of hysteria was amply justified by the way in which she got well, that as soon as her individual whim was gratified, just so soon improvement took place; that at any time she might have gotten rid of the hysteric anorexia and vomiting if she had only made up her mind to do so.

This incident was lost to my consciousness for a number of years until one day in Mercy Hospital some years ago during the life of Dr. J. B. Murphy we had together a case which showed a terrible amount of vomiting. The vomiting was pernicious and gastric lavage was of no avail. It looked as though the patient was going to die after a gastrojejunostomy. At that time we both had had our attention to some extent turned to acetonemia, which was then being very vaguely hinted at, and had the urine examined for acetone. It was present. Like a flash the memory of the case of the girl with the hysteric vomiting came to my mind, and I proposed that we give the patient cider, making the point that the cider contained a good deal of fructose, a fruit sugar, which was a natural glucose, and mentioning this case which I had not thought of for many years. The cider was obtained from a nearby grocery store and given to the patient. The vomiting was controlled and the patient got well. From that time on we began using cider more or less increasingly, believing, however, that cider was not the essential thing, but rather the fruit sugar or fructose, so that later we made use of strained honey, orange juice, which contains less

sugar than other juices, and grape juice. Pure grape juice not prepared with cane-sugar is very excellent, but hard to obtain. Ordinary grape juice does not agree with most patients because of the cane-sugar which it contains. Whatever article is used does not matter provided there is put into the stomach of these patients or into the colon by proctoclysis the dextroses which they are so much in need of. Levulose is useless.

For five years there has not been a death in Mercy Hospital on any service from gastrojejunostomy. This is a splendid record and one that cannot be improved upon anywhere. Practically the only cause of death from gastrojejunostomy at present is acidosis because the technic is so far perfected that there is no danger of losing the patient from any other cause. If the acidosis is controlled as it can be simply and yet satisfactorily the convalescence of the patient becomes a thing of joy rather than a thing of extreme anxiety.



## CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

### THE RECOGNITION AND TREATMENT OF DIFFERENT TYPES OF AURICULAR FIBRILLATION

**Frequency with which Auricular Fibrillation is Now Recognized. Two Series of Phenomena Occurring in this Condition. Auricular Fibrillation Perhaps the Most Persistent Irregularity of the Human Heart. Results of Digitalis Treatment. Rôle of Quinidin Success in Management Dependent on Differentiation of Various Types and Treatment of Underlying Disease Process.**

THE clinical condition, auricular fibrillation, is simple and easily understood, the cardiac mechanism responsible for its production not difficult to appreciate. Furthermore, the condition is not infrequent, as it is met with in private, hospital, and dispensary practice, and is important to recognize, as much can be done for its control in a practical, therapeutic way. The frequency of its recognition increases parallel with the attention and consideration given it, recent cardiac literature bringing testimony to this point.<sup>1</sup> There are several varieties, as it occurs under varying conditions, and its complete recognition includes the differentiation of these varieties, and in some instances the diagnosis of the underlying disease process. Finally, its separation from other types of irregularity must be made. Both the recognition and differentiation can usually be made by the simplest clinical means—the stethoscope, one's fingers, and one's head, without the use of special instruments of precision such as the polygraph and the electrocardiograph.

If one inspects the beating of a normal rabbit's heart after the chest wall has been removed one sees the regularly recurring

<sup>1</sup> During the past five years the references on auricular fibrillation in the Quarterly Cumulative Index have increased from seven articles in 1916 to twenty-eight in 1921.

sharp contraction of the auricle followed, each one, by a contraction of the ventricle. The contraction of the auricle is seen as a sharp, quick "flick." If the auricle is thrown into fibrillation (by an induction current), this normal "flick" disappears, the auricle appears paralyzed or resting, as if in diastole. Closer inspection, however, shows that it is not entirely quiescent, but that its contraction is composed of multiple small fibrillary twitchings of individual muscle-fibers. In other words, auricular fibrillation of the human heart is "a condition in which the auricles fail to contract *en masse*, the muscle activity consisting only of fibrillary twitchings; the normal and regular impulses transmitted to the ventricle are absent, while rapid and irregular impulses produced in the auricle replace them and produce gross irregularity of the ventricular action" (Lewis).

The literature on fibrillation contains a large number of synonyms for this interesting condition, which terms are likely to be confusing unless their significance is appreciated. Under the terms "delirium cordis" and "complete irregularity of the pulse" Bouilland, in his "Les Maladies du Cœur," in 1836, and Nothnagel in 1878, described what today we recognize as auricular fibrillation, the latter describing a case which we would probably identify as belonging to the paroxysmal type. Riegel in 1898 discusses it under the term "mitral pulse" from "injury to cardiac muscle." The new era of physiologic heart studies, particularly studies in the arrhythmias, dates from the work of McWilliams, Gaskell, and Engelmann on the frog's heart, with the resultant fruitful myogenic theory, the work of Cushing on the vertebrate heart and of Wenkebach on the human heart (1899). H. E. Hering in 1903 introduced the term "pulsus irregularis perpetuus," and D. Gerhard in 1910 "arrhythmia perpetua." Mackenzie in 1902 with Wenkebach spoke of it as "nodal rhythm." Rothberger and Winterberg in 1909 first showed, by electrocardiographic means, the relationship between arrhythmia perpetua of man and fibrillation of the auricles of vertebrate animals, although the suggestion had been made three years earlier by Cushing and Edmunds.



## RECOGNITION

As was intimated above, there are several clinical varieties of fibrillation, but before discussing these it might be profitable to picture the type one most frequently meets and which is most easily recognized, namely, the one associated with heart failure. Moreover, this type is perhaps of most importance to recognize clinically because of the brilliant results which can often be obtained by accurate therapeutic management. The patient of this type, a man or woman in the fortieth or fiftieth decade of life, enters the dispensary or hospital or is seen in private practice evidencing the easily recognized findings of heart failure. The symptoms of this group in the order of their frequency are perhaps breathlessness on exertion (amounting at times to orthopnea), precordial or epigastric pain (not often anginal), swollen legs and abdomen, varying degrees of cyanosis, palpitation, weakness, and cough. On feeling the wrist the pulse is found to be rapid, weak, and markedly irregular, the rate difficult to count, varying at the wrist from 60 to 120 and at the apex from 90 to 170, the difference between them representing lost beats or the so-called "pulse deficit." On auscultation the beating is found to be rapid and tumultuous, corresponding to the patient's complaint of palpitation. The irregularity of the pulse is both of rhythm and of force, the former being evidenced by the irregularity in the spacing of individual beats, the latter showing variation in the strength or force of separate beats, which latter findings may be easily confirmed by variations in the blood-pressure levels of these beats.

An important, easily demonstrated fact is that the irregularity is made markedly worse by effort or exercise. This may be elicited by asking the patient to sit up and lie down several times in succession, until beginning evidence of distress, dyspnea, or fatigue are manifest. This effort test markedly increases the rate, tumultuousness, and irregularity of the pulse, and this easily applied clinical test serves as a differentiation from the most commonly confused condition, premature contractions (extrasystoles). In this latter condition several quick changes

from the recumbent to the sitting posture tend to *abolish* the premature beats. Conversely, upon rest, as the pulse slows down, fibrillation *decreases*, while extrasystoles *increase*. Likewise, with other measures which augment the pulse-rate, such as fever or amyl nitrite, the irregularity of fibrillation becomes worse, while that of extrasystoles tends to disappear.

Auricular fibrillation gives rise to two series of phenomena: first, those due to paralysis or fibrillation of the auricle; second, those due to disordered action of the ventricle. Discussing the latter first, if the ventricle is beating *rapidly* the pulse is turbulent, varies in rate from 100 to 160, and is easily recognized as the irregular, rapid pulse of fibrillation. However, when the ventricle is beating *slowly*, from 80 to 100, the pulse is much less turbulent and, on hasty examination, may appear to be perfectly regular. The recognition of the irregularity in this group of slowly beating hearts is more difficult, and may necessitate the aid of the polygraph or the electrocardiograph for its certain diagnosis. On auscultation, the heart sounds due to ventricular action are modified, they vary in intensity corresponding to the variation in force of the radial pulse-beats, the second sound being often lost when the pulse-beat is lost.

Turning now to the auricular signs, in fibrillation the commonest clinical change is in the presystolic murmur of a mitral stenosis, the presystolic element of which, when the heart goes into fibrillation, disappears or is lost, a protodiastolic or mid-diastolic murmur taking its place. In the event that fibrillation ceases, the presystolic (auricular systolic) murmur may again be heard, with the resumption of sinus rhythm.

Auricular fibrillation is perhaps the most frequent, persistent irregularity of the human heart. It has been estimated that 50 per cent. of all types of irregularity belong to this group, while 60 to 70 per cent. of all cases of heart failure entering a general hospital will be found to have auricular fibrillation (Lewis). In general, it may be anticipated that cardiac irregularities in which the rate remains around 120 per minute or over, irregularities accompanying heart failure, irregularities which become worse with fever, exercise or amyl nitrite, that

all of these are, in the majority of instances, cases of auricular fibrillation.

For the most part the symptoms of patients with auricular fibrillation are due to the degenerated and failing heart muscle, but there are some symptoms which are due to the specific effects of fibrillation itself. Of these may be mentioned the occasional spells of fluttering felt in the chest or neck; the suggestive consciousness of irregular heart action; the tendency to dyspnea, exhaustion, and other symptoms of overtaxation; the peculiar freedom from angina. These symptoms are probably due to the actual turbulence, embarrassment, and rapid action of ventricular movement, not from fibrillation of the auricle and not primarily from heart muscle failure. A brilliant example of the effects and relief of these symptoms due to disordered ventricular action was recently afforded by a ward patient, entering the hospital with marked cyanosis, orthopnea, palpitation, etc., and a rapid type auricular fibrillation. The prompt (within twenty-four hours) restoration of sinus rhythm by quinidin was accompanied by the most dramatic relief, practically complete, in the above-mentioned symptom.

#### CLASSIFICATION

The classification of auricular fibrillation may be made in one of two ways: first, as to cause (etiologic classification), and second, as to permanency (transient, paroxysmal, or permanent). The first class may be further subdivided into the rheumatic and non-rheumatic groups. Approximately 66 per cent. of all fibrillators belong to the so-called rheumatic group, this group occurring preponderantly in women between the fifteenth and fortieth years and in women suffering from mitral stenosis (52 per cent.). An example of this type is the following case in a young girl, E. L., aged sixteen years, suffering with a long-standing case of mitral stenosis and regurgitation following repeated attacks of acute rheumatic fever. This child, whose pulse had always been perfectly regular, suddenly developed an acute attack of nausea, vomiting and syncope, and was brought into the hospital with marked signs of heart failure

and a rapid and grossly irregular pulse. Her first electrocardiogram taken December 27, 1920 (Fig. 302) before digitalis was administered shows the typical curve of auricular fibrillation with a ventricular rate of 130. Later curves show clearly the beneficial effect of digitalis; within four days of the administration of 12 c.c. tincture of digitalis the ventricular rate dropped to 63. Four days later, on January 3d, after 16 c.c. of the tincture, the ventricular rate had dropped to 54, with signs of mild

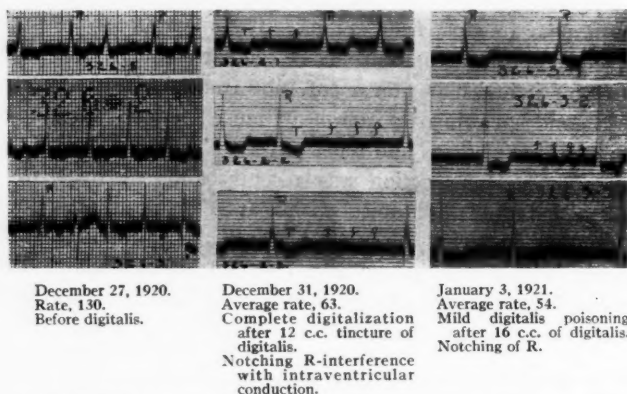


Fig. 302.—E. L. Age sixteen. Rheumatic endocarditis and myocarditis. Mitral stenosis and regurgitation. Case of long-standing mitral stenosis with sudden attack of heart failure and auricular fibrillation, ushered in with nausea, vomiting, syncope; later, signs of decompensation. Shows slowing effect of digitalis, inversion of T in all leads, and disturbed intraventricular conduction.

digitalis-poisoning. Since this time she has been able to resume her work with intermittent small amounts of digitalis.

The non-rheumatic group occurs most frequently in men between the forty-fifth and seventieth years, in patients suffering from cardiovascular-renal conditions, myocardial degeneration with heart failure, generalized arteriosclerosis. Mr. W. E. P., a building contractor aged fifty-two years, suffering for years with recurrent attacks of dyspnea and cyanosis, is a typical example of this group. His pulse had been permanently irregular (fibrillation) for ten years. He was a nervous, energetic, diffi-

cultly controlled patient, only willing to accept treatment and bed-rest when severe dyspnea and orthopnea compelled him to do so. His blood-pressure ranged from 170 to 210. His curves are shown in Fig. 303. Digitalis was able to hold his ventricular rate down to 90, but at no time was he willing to accept prolonged bed-rest and adequate treatment. His dramatic death, occurring during sleep at night, prostrated his wife, but was not wholly unexpected by his attending physician, although there was nothing in his electrocardiogram which predicted his sudden demise.

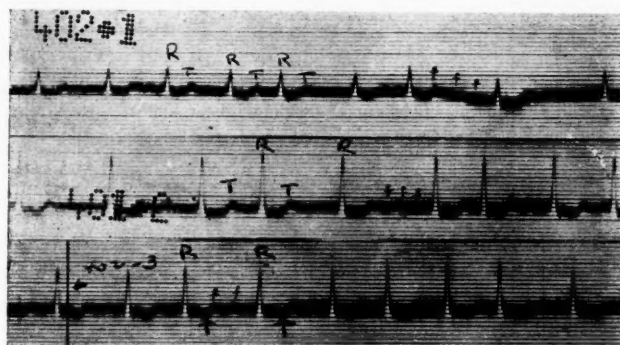
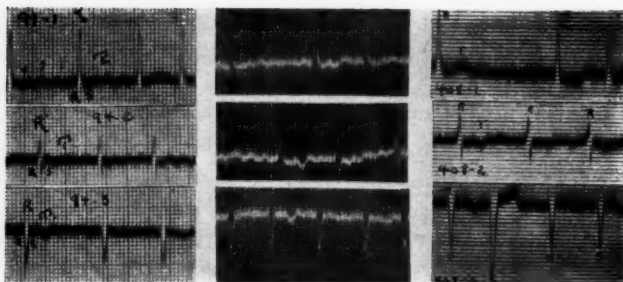


Fig. 303.—W. E. P. Age fifty-two. Average rate, 88. Chronic myocardial disease. Hypertension. Auricular fibrillation. Marked dyspnea, cyanosis, orthopnea. Sudden death while asleep.

Patients in this non-rheumatic group are often seen grouped in families, as are other types of the cardiorenal-vascular groups (hereditary arteriosclerosis). For example I, as others, have seen two or three members of the same family suffering and eventually dying from angina pectoris; several members of another family die from cerebral thromboses and apoplexy; another group gives evidence predominatingly of uremic complications. The 2 following cases, brothers, were members of a cardiovascular familial arteriosclerosis, with outspoken signs of auricular fibrillation. Figure 304 shows the curves of the younger, J. M. W., aged sixty, ill for three years with progres-

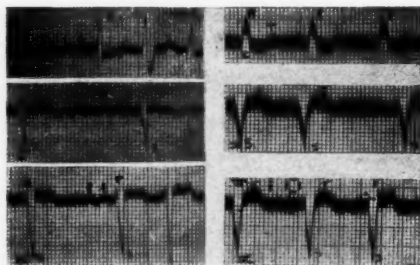


April, 1919.  
Auricular fibrillation.  
Left ventricular preponderance.  
No evidence of heart failure.  
Blood-pressure 170/110.

October, 1919.  
Beginning heart failure.  
Moderate amounts of digitalis.

February 5, 1921.  
Nine months after recurrent severe cerebral thromboses, three months before death.  
Blood-pressure 220/130.  
Ventricular extrasystoles.

Fig. 304.—Mr. J. M. W. Age sixty. Familial arteriosclerosis. Chronic myocarditis. Auricular fibrillation. Beginning heart failure. Recurrent attacks of cerebral hemorrhage and thrombosis.



Taken February 28, 1917.  
Average rate, 87.  
Auricular fibrillation.  
Ventricular extrasystole.  
Left ventricular preponderance.  
Recovery from heart failure.

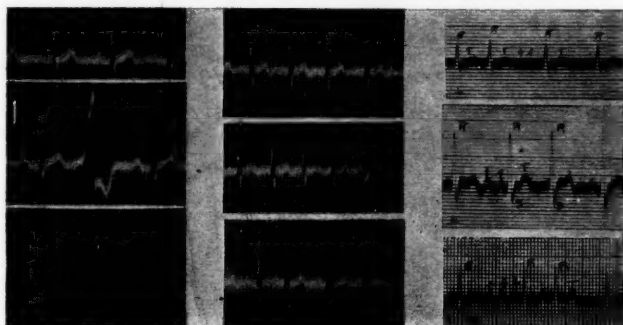
Taken April 12, 1919.  
Two years after heart failure.  
Average rate, 82.  
Evidence of disturbed intraventricular conduction.

Fig. 305.—Mr. E. W. Age sixty-six years at death in 1920. Auricular fibrillation with ventricular extrasystoles in chronic cardiovascular disease with recurring heart failure, disturbed intraventricular conduction with sudden death from probable ventricular fibrillation due to coronary embolism or thrombosis in a previously diseased coronary system.

sive heart failure, increasing blood-pressure, and recurrent attacks of cerebral thromboses. The older, E. W., aged sixty-

six, with practically the same disease picture, died a more sudden death from probably ventricular fibrillation (Fig. 305).

From the standpoint of the second class, permanency, the following five subdivisions are recognized: 1. Transient fibrillation occurring in the course of acute infectious diseases or with various acute fevers, pneumonia, acute rheumatic fever, typhoid, paratyphoid, diphtheria, severe tonsillitis, acute cholecystitis, acute appendicitis, scarlet fever, acute endocarditis, acute pericarditis. A striking example of this group is that of H. R.,



Taken before attack.  
Average rate, 96.  
Sinus rhythm.  
Ventricular extrasystole.

Taken during attack.  
Average rate, 140.  
Auricular fibrillation.

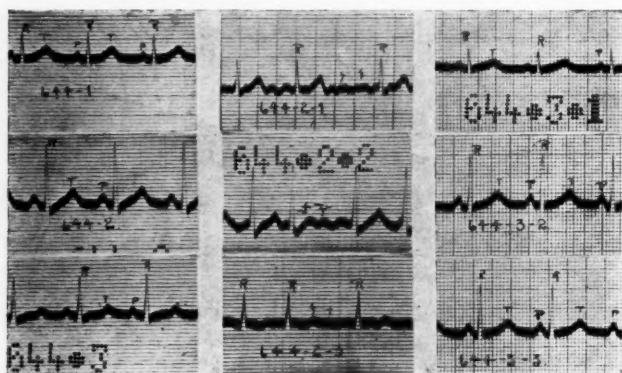
Taken during attack.  
Average rate, 120.  
Auricular fibrillation.

Fig. 306.—H. R. Age fifty-two. Physician. Postinfectious chronic myocarditis. Auricular fibrillation. Ventricular extrasystole. Four attacks of recurrent paroxysmal fibrillation covering a period of thirty years. First attack during acute paratyphoid infection, two while operating, one while swimming. Spontaneous occurrence and remission.

a physician aged fifty-two, who has experienced four attacks of recurrent paroxysmal auricular fibrillation during a period of thirty years. His first attack of irregularity occurred during his internship while suffering from an acute paratyphoid infection. Both his second as well as his fourth attack occurred while operating, at which time, while in the midst of a tedious, difficult surgical procedure, while bending over the operating table, he became suddenly faint, dizzy and nauseated, necessitating his sitting down and discontinuing the operation. Upon



feeling his pulse, which had previously been entirely regular, he found it to be extremely rapid and irregular. The fourth attack, curves of which are shown in Fig. 306, lasted five days and was associated with quite severe precordial distress, with, at times, radiation to the left shoulder and left arm—a rather striking picture of angina. This last attack, within a week's time, ceased as abruptly as it came with bed-rest and an ice-bag to his precordium. The third attack occurred while he was in swimming, the sudden associated weakness and dizziness



October 19, 1921.  
Rate, 84.  
Normal cardiac mechanism.

October 23, 1921.  
Rate, 150.  
Auricular fibrillation acute.  
No quinidin.

October 26, 1921.  
Rate, 83.  
Sinus rhythm after 1.6 gm.  
of quinidin.

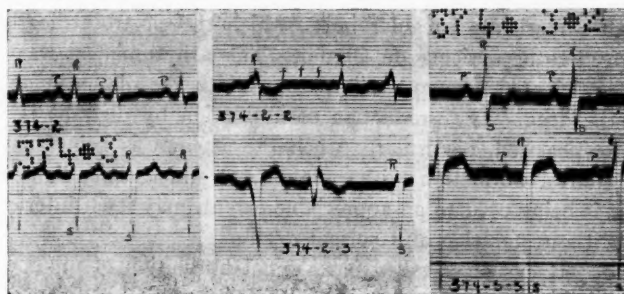
Fig. 307.—Mr. J. K. Age fifty-eight years. Hyperthyroidism. B. M. R. 50.4+. Generalized arteriosclerosis. Transient auricular fibrillation controlled by quinidin.

making it appear doubtful if he could reach the shore. Following the last attack he vacationed for three months in California and has since then materially reduced his work, with, up to the present time, no recurrence of his trouble.

2. Transient fibrillation occurring in the course of surgical anesthesia or following shortly after surgical operations. 3. Fibrillation from disturbance of the thyroid gland, particularly in Basedow's disease. The fibrillation somewhat paralleling the thyroid intoxication. Cases of this variety are extremely interesting, and



it has been my privilege in the past few months to have seen several striking examples of this type. Figure 307 shows several curves from a Basedow patient, an insurance man of fifty-eight years, suffering with general weakness, anorexia, trembling, nervousness, loss of weight and diarrhea, with a basal metabolic rate of 54+. Upon entrance to the hospital this patient's pulse was regular and normal, with a rate of 84. Suddenly, four days after admission, he developed an acute delirium cordis, with a ventricular rate of 150. Within three days after the administration of 1.6



December 30, 1920.  
Advanced heart failure.  
No digitalis.  
Ventricular extrasystoles.  
Rate, 100.

January 2, 1921.  
After 16 c.c. of digitalis.  
Mild digitalis poisoning.  
Auricular fibrillation.  
Rate, 90.  
Ventricular extrasystoles.

January 13, 1921.  
Digitalis discontinued.  
Compensation restored.  
Normal cardiac mechanism.  
Rate, 65.

Fig. 308.—Mr. N. H. Age fifty-two. Transient auricular fibrillation from digitalis. Advanced heart failure. Left preponderance. Chronic myocarditis.

gm. quinidin his irregularity gave way to a sinus rhythm. During the six months that has elapsed since he was under observation he has had many remissions of fibrillation, with subsequent restoration of sinus rhythm, but has now, as the result of a marked and permanent improvement in his thyroid condition, apparently permanently, a regular, slow pulse.<sup>1</sup>

4. Fibrillation brought on by toxic amounts of digitalis in association with other signs of digitalis-poisoning, namely,

<sup>1</sup> This case has previously been reported in the paper on "Effects of the Administration of Quinidin Sulphate in Auricular Fibrillation," W. W. Hamburger, Jour. Amer. Med. Assoc., December 3, 1921, lxxvii, 1797.

digitalis heart-block, digitalis nausea and vomiting, digitalis headache, vertigo, tinnitus, etc., which disappear promptly with the cessation of the drug. Curiously enough, the drug which is of supreme value in the treatment of auricular fibrillation, will itself, under certain conditions, produce auricular fibrillation in an otherwise regular pulse. Figure 308 shows an example of such curves. This patient, a merchant fifty-two years of age, suffering with advanced heart failure, with a regular, though rapid heart, experienced, after the administration of 16 c.c. tincture of digitalis, signs of mild digitalis-poisoning with a markedly irregular pulse, which the electrocardiogram proved to be auricular fibrillation. This arrhythmia, as was to be anticipated, promptly disappeared with the discontinuance of the drug. The production of fibrillation by digitalis is to be emphasized, particularly when the attempt is being made to control fibrillation with quinidin, at which time digitalis, if it is being used to an appreciable extent, may itself interfere with the restoration of a normal rhythm.

5. Attacks of so-called paroxysmal fibrillation of unknown origin in either men or women, probably often heralding the onset of temporary or permanent heart muscle failure; often associated with paroxysmal dyspnea, periodic vertigo and nausea, attacks usually disappearing promptly with rest in bed without any special form of treatment; often resulting (as many believe), after many recurrences, in permanent fibrillation.

#### PROGNOSIS

While in a given case the exact mechanism and significance of the onset of an attack of auricular fibrillation may not be clear, it is generally agreed at present that fibrillation is an expression of structural heart muscle changes, and that its onset is evidence of beginning or advanced heart muscle failure. The onset in most cases heralds and is synchronous with heart muscle failure, either temporary or permanent, as few individuals survive permanent fibrillation more than ten to fifteen years. It is obvious that a persistent ventricular rate of 120 is serious, that a persistent rate of 140 may endanger the person's life

within a few months, while an untreated rate of 160 or more may terminate in a few weeks. Fortunately, these higher rates can usually be controlled by digitalis. Therefore the reaction to digitalis treatment is of prime importance, and it may be stated that the prognosis depends not so much on the *heart rate first observed*, as on the heart rate remaining after treatment has been instituted.

Other important factors in the prognosis are, of course, the general appearance and condition of the patient, for although the heart rate may remain slow with little or no digitalis, the signs of advanced heart failure may persist or even increase, in which event the outlook is obviously unfavorable. The prognosis then is the same as in other cardiac affections, depending upon the integrity and reserve of the ventricular heart muscle, much more than on the regularity or irregularity of the auricle. P. D. White has emphasized the seriousness of fibrillation complicated by aberrant ventricular complexes and ectopic ventricular contractions, believing that such cases are almost as fatal as patients suffering with pulsus alternans. F. A. Willius, analyzing the results in 500 cases of fibrillation, concludes that the mortality attending auricular fibrillation doubles and in some instances trebles that occurring in similar types of heart disease not complicated by this arrhythmia.

#### TREATMENT

There is no other serious cardiac disorder so speedily and brilliantly benefited as the well-managed case of auricular fibrillation, as it is this condition, above all others, to which drugs of the digitalis series owe their well-founded reputation. The most important guide in treatment is the heart-rate, as, irrespective of the presence or absence of heart failure, an apex rate of 100 or over is an absolute indication for digitalis. The drug acts practically as a specific, impeding the passage of the rapid, haphazard impulses from auricle or ventricle, thus reducing the ventricular rate.

The results of digitalis treatment may be classified under three headings: 1. Those cases in which the reaction is perma-

nent, the rate remaining slow, although digitalis is omitted. 2. The reaction is permanent only when small doses are subsequently administered. 3. The reaction may be permanent only with the administration of relatively high amounts of the drug.

**Routine Management.**—If the patient's distress is not extremely urgent and if he has had no digitalis within ten to fourteen days, my practice is to put him on 15 to 20 minims of a good tincture of digitalis three to four times a day by mouth, continuing this dosage until a fall of rate to 80 is obtained or until signs of intoxication occur. This necessitates, on the average, from 6 to 8 to 10 drams of the tincture over a period of seven to ten days. Continuation of this amount is not without untoward dangers, and signs of intoxication must constantly be looked for, especially a sudden drop of rate, the occurrence of coupled beats, nausea, vomiting, etc. If the need is more urgent, 30 minims four times a day may be given, or, if the tincture is not well tolerated by mouth even after considerable dilution with water and administration after meals, strophanthin may be given in doses of  $\frac{1}{250}$  grain in small amounts of sterile salt solution, intravenously, two or three times a day at two- to four-hour intervals.

Aside from the administration of digitalis in auricular fibrillation there has, in most recent time, been added a most brilliant therapeutic aid in the treatment of these patients, namely, quinidin. Although the exact rôle and position of the quinidin treatment of fibrillation is as yet not fully established, it may safely be said that one is not justified in handling a case of this type of irregularity at the present time without at least considering carefully the possibilities of quinidin treatment. Without going into this phase of the subject thoroughly at this time, as it is not within the province of this clinic, the following statements may be considered justified in the light of our present knowledge: Unless there is some specific contraindication, quinidin should be administered in cases of auricular fibrillation of short duration; in cases not associated with advanced, severe, or recurrent heart failure; in cases not com-

plicated by other forms of irregularity—ectopic ventricular contractions, aberrant ventricular complexes; in cases without idiosyncrasy to quinin or its derivatives. It is probably especially useful in acute fibrillation as distinguished from chronic fibrillation, particularly in those cases developing immediately following some acute infectious disease, during the course of a Basedow, following surgical operation. It should be administered with extreme care, if possible in a hospital with electrocardiographic

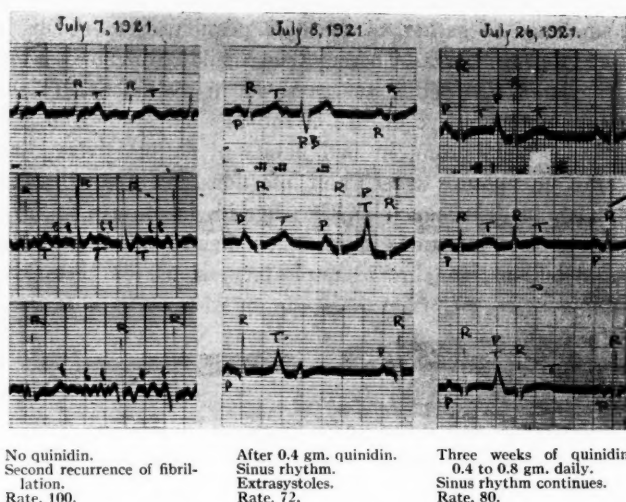


Fig. 309.—J. M. Age forty-three. Mitral stenosis. Auricular fibrillation. Restoration of normal (sinus) rhythm following administration of 0.4 gm. quinidin sulphate.

controls, always with a preliminary idiosyncrasy test, probably with preliminary digitalization in the presence of heart failure, in minimum dosage working up to a larger amount if the smaller quantities are ineffectual, under conditions of bed-rest and competent medical and nursing control of the patient. Figure 309 shows the results of a patient undergoing quinidin administration. Within twenty-four hours after the administration of 6 grains of the drug in this patient, a man forty-three years

of age suffering with mitral stenosis, a normal rhythm had been restored. Finally, it should never be forgotten that even with the controls listed above, there have already been reported several instances of collapse, syncope, respiratory failure, paralysis, and even death in the administration of this drug.

Lastly, in the event that quinidin does not control fibrillation permanently, digitalis should be given in such a way and the patient's life so regulated that heart failure may be prevented or postponed. During this period no stone should be left unturned to gather all the data possible as to the basic cause of the fibrillation. Therapy then should be directed toward its amelioration, whether the cause lie in malfunction of the kidneys, in progressive disease of the vascular system, in perversion of metabolism, in bad habits of eating, living, and working, in exposure to recurrent infections, in chronic diseases (syphilis), in disturbances of the thyroid, in absorption from chronic foci of infection, in nicotine, caffeine, alcohol, or other chronic poisons, in chronic respiratory, sinus, or rheumatic infections.

CLINIC OF DRS. PHIL A. DALY AND  
SOLOMON STROUSE

CHICAGO LYING-IN HOSPITAL AND DISPENSARY

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HEART DISEASE AND PREGNANCY

**Management of Cardiac Complications of Pregnancy Should Not Be Attempted on a Basis of Generalization of Heart Lesions. Each Patient Should Be Individualized By Observation By Both Obstetrician and Internist. Presentation of 4 Patients Illustrating the Results of this Plan of Management.**

BEFORE presenting to you the patients whose stories form the basis of this clinic we wish to call attention to the somewhat unusual situation of presenting medical cases from an obstetric hospital. For the past few years Dr. DeLee and one of us (S. S.) have together studied all pregnant women at the Chicago Lying-in Hospital who presented any medical complications. During the past year a prenatal and postnatal dispensary clinic has been held weekly, at which all pregnant women with medical complications have been rounded up for observation (P. A. D.). The dispensary is a direct part of the hospital, and at any time hospital observation was necessary the patient was immediately sent in. It surprised all of us to find how large the material was, and how great an opportunity was furnished for team work, especially in defining therapeutic indications.

It seems natural that overspecialization in medical practice has the evil side of overdevelopment of one point of view of the patient at the expense of the whole patient. If the facilities of large medical institutions are to be utilized to their fullest capacity for the good of the patient, the material in such institutions should receive well-rounded study from every point of view.

As an example of the type of work we are trying to do we present today some patients with heart disease in pregnancy.

You will note that our remarks concerning these patients are entirely clinical, and that with the exception of the sphygmomanometer no instruments of precision were employed in the study of the cases. While the absence of data accumulated by x-ray or electrocardiogram may seem significant, after all, judgment as to therapeutic procedures in pregnant women with heart disease must be mainly clinical. While it would be of unquestioned scientific and academic interest to procure such information, we do not feel that our conclusions are embarrassed by their absence.

Generalizations concerning the significance of cardiac complications in pregnancy are common. One group holds conditions short of actual *failure* too insignificant for consideration; the other regards *any* lesion as too serious to be managed by means other than operative interruption of pregnancy and grave warnings against future pregnancies. It seems possible that neither extreme gives the best results. Basing conclusions on diagnosis alone is not the point of view which gives best therapeutic results. The individual cases should be observed by both obstetrician and internist, the serious differentiated from the mild, and therapy advised entirely according to the individual possibilities.

Our aim is twofold: First, of course, to carry every pregnancy to viability, if possible, without endangering the life of the mother; second, to arrive at a decision as to advisability of subsequent pregnancies.

In formulating a plan of management we must try to answer several questions:

1. What is the etiology, and are there associated vascular or renal lesions?
2. Will the heart actually fail?
3. Is the pregnancy increasing the *damage* to the heart?
4. Can we offset the increasing burden of pregnancy by decreasing the patient's voluntary effort, or must we interrupt the pregnancy?



5. Will the interruption injure the patient as much as continuance of pregnancy?

6. Will the interruption before viability of the fetus save the mother enough injury to warrant destruction of the child, or can she carry the fetus until there is at least a possibility that it will live?

7. How will subsequent pregnancies affect her?

8. What are the signs which indicate interference?

9. Should we eliminate the effort of parturition entirely or allow complete dilatation and eliminate the second stage?

In addition to these points we must consider the parents' desire for at least one living child, the religious feelings of the parents, and the difference in attitude toward the first and, for example, fifth or sixth child. Some of these points are not strictly medical, but at times the desire of a woman for at least one child is so strong that she is willing to run the risk of the supreme sacrifice. In such instances as in those in which religious feeling offers an unanswerable argument against scientific advice the physicians are compelled to accept the word of the mother as final and do everything humanly possible to carry pregnancy to the point where the child is positively viable, then interfere.

A diagnosis does not make a prognosis. Many women who have mitral stenosis or aortic insufficiency go about their daily tasks, and through pregnancies, unhampered by any cardiac distress, while others with similar lesions (from a diagnostic standpoint) are confined to bed, experiencing considerable discomfort even when at absolute rest. It is true that most women, regardless of lesion, will go through pregnancy, delivering either by premature labor or spontaneously at term, and live; Sir James Mackenzie states that very seldom will a woman die from cardiac failure during labor unless grossly mismanaged. It is true also that the prevention of marriages or pregnancies, or the emptying of uteri in the early weeks will eliminate any possibility of the pregnancy aggravating or increasing the cardiac impairment, but is either course justifiable in every case? Cannot co-operation of internist and obstetrician

evolve a more rational and conservative plan whereby greater good may result to all parties concerned, even to the one who has no voice in the procedures, though a very interested party—the baby?

A normal heart in non-pregnant woman is the same as in the pregnant, a diseased heart in non-pregnant is the same as in the pregnant. But conditions are different; the heart of the pregnant woman has additional burdens and handicaps in working conditions which impair, to greater or less degree, its efficiency. The efficiency of the heart depends, of course, upon the health or disease of its musculature, compared with which the anatomic lesions present is insignificant. The degree of adaptability of the heart is measured by the reserve force of the myocardium. If the heart muscle is good, which means that the reserve force of the heart is sufficient to adapt itself to added work, then the valvular lesion which may be present makes little difference in the ability of that heart.

Murmurs are the greatest source of confusion and mistake. They may, but not always, lead to a diagnosis, but aside from statistical and theoretic probabilities, assist but little in prognosis; of themselves they afford but a vague and uncertain idea as to the degree of impairment. We know that mitral stenosis and aortic regurgitation are the most serious lesions encountered in pregnant women, but every presystolic mitral murmur does not necessitate a grave prognosis. Nor do diseased hearts presenting the same lesion and similar physical findings (objectively) necessarily mean the same prognosis.

Something of the truth of these statements we will attempt to demonstrate by 4 patients who have been chosen from a series of cardiac complications of pregnancies. Two are mitral stenoses. Two are aortic insufficiencies. One of each pair has been delivered by cesarean section and at the same time sterilized, the others delivered spontaneously, with such obstetric assistance as was deemed necessary, and were not sterilized.

**Case I.**—Mrs. H., No. 19,258, twenty-eight years of age, para I.

*History.*—She has had “heart trouble” since childhood and also chronic recurrent arthritis (multiple). There is no history of complete cardiac failure at any time, but always some breathlessness and edema of feet. When first seen she complained of rather severe dyspnea and swelling of extremities, the increase of subjective symptoms becoming very noticeable about the fifth month of pregnancy. She was unable to do her housework without considerable distress and had difficulty in breathing when lying flat in bed.

*Findings.*—Patient is a small woman, rather anemic, with a diffusely enlarged, soft thyroid, but no toxic signs. Lungs are clear. Heart: Left border of dulness is 10.5 cm. from midsternum (in this woman anterior axillary line). Right border is 3.5 cm. to right. There is a palpable pulmonic second and apical presystolic thrill, thrill followed by “shock” of apical impulse. There is a presystolic murmur, crescendo type, at apex; systolic and diastolic apical murmurs are also present. Liver is two fingers below arch; no pulsation is present. Blood-pressure is 135/80, pulse 90 and upward, temperature 98.6° F. Small joints of hands are moderately deformed by arthritis. Diagnosis: Mitral stenosis and insufficiency, chronic arthritis.

Patient was observed for several weeks while at home and with activities somewhat limited. Symptoms unabated. Next as complete rest as was possible under home conditions, still symptoms continued unchanged, showing that her cardiac reserve was not equal to even very moderate exertion. Patient was sent to hospital, where under complete bed rest and digitalis she became quite comfortable, but any exertion tended to bring about dyspnea.

Cesarean section with sterilization was advised because of very apparent lack of cardiac reserve in addition to the organic lesions present. Operation was performed under ether anesthesia and a live baby delivered. Patient passed through operation and postoperative period without trouble. After ten days very gradually increasing exercise was permitted and the patient left the hospital in good condition. She returns today, some five months after operation. We find now that

she has about the same degree of breathlessness as she experienced previous to her pregnancy, does her housework with no marked dyspnea, but does tire rather easily. Physical findings about the same, a slightly decreased breadth of cardiac dullness, and a less audible diastolic murmur. Rate 84. No edema. Condition practically the same as before pregnancy. Baby is living and well.

**Case II.**—Mrs. L., No. 21,405, twenty-seven years, para II.

*History.*—She becomes easily tired with moderate exertion; this became noticeable soon after onset of pregnancy, first pregnancy normal and with no similar symptoms. There is no history of onset or acute infections or arthritis obtainable. She has moderate edema at times. She is six months pregnant.

*Findings.*—The patient is a large, heavy woman. Head and neck are negative. Lungs are clear. Heart: Left border cardiac dullness is 11 cm. from midsternum (well inside anterior axillary line). Right border of cardiac dullness is 3 cm. from midsternum. There is a short presystolic thrill palpable at apex, with presystolic and systolic murmurs at apex. Pulmonic second sound is much accentuated. Abdomen is negative except for pregnant uterus. Extremities are negative. Diagnosis: Mitral stenosis and insufficiency.

Observed under home conditions with only moderately reduced activities. Progressed quite favorably, breathlessness becoming very gradually more easily induced as pregnancy progressed and uterus increased in size. Abdomen became very large, large baby, and patient entered hospital only a few days before delivery. Labor occurred spontaneously, an 11-pound baby was delivered with midforceps, with patient under ether anesthesia.

Convalescence uneventful, and today patient has very little complaint, very moderate degree of breathlessness upon exertion. She is doing her own housework. Physical findings are identical as far as one can make out by physical examination except that the apex is lower and possibly closer to median line due to emptying of the uterus.

What were the indications for the wide difference in treatment of these two patients?

The final decision for cesarean section in one patient and against in the other rested upon the cardiac reserve force. In the first the reserve was sadly lacking, was barely capable of maintaining efficient circulation in the non-pregnant state with the woman doing a moderate amount of work. The etiologic basis in Case I was apparently infective, she still has recurrences of acute arthritis, and evidence of long-standing chronic process. The cardiac reserve was so limited that we feared failure of that heart under additional strain. The pregnancy had definitely aggravated the cardiac symptomatology, and the increase could not be offset by decrease in voluntary effort. Operation was considered less injurious than the strain of spontaneous delivery and allowed also the opportunity of sterilization. The fetus was viable, she will have one living child. Further pregnancies, if undertaken, would certainly aggravate her condition as much or more than this, for the ultimate prognosis, even without subsequent pregnancies, is bad; with the additional burdens and dangers of future pregnancies the outlook would be very much worse. If the lesion as it now is progresses no further, if the stenosis does not increase (and all stenoses tend to increase), the present state of health may be maintained, by careful living, for some time. At any rate, by surgical delivery a deal of strain has been spared that myocardium. Sterilization has removed a source of future burden, and possibly the life expectancy has been increased by a margin, which, though it may not be great, is rather precious to the family concerned.

The second patient, on the other hand, presents no evidence of marked impairment of reserve power. In the non-pregnant state the routine duties of her life are accomplished very comfortably, her enjoyment of life is not hindered by cardiac inefficiency. In the pregnant state the physical exertion of household duties is supplanted by the extra burden of pregnancy, either she handles very well, both together are a little beyond the adaptability of that heart, though causing no failure or

marked distress. So that by restricting her voluntary efforts pregnancy is borne rather well. There is no present or recent active infection pointing to an infective focus. We have no basis for fearing failure, there seems to be no increasing damage to the heart, and the very moderately aggravated symptomatology is readily diminished by decreasing voluntary effort. Not fearing failure and not wishing to sterilize, there is no contraindication to spontaneous labor. The physical findings of the second woman are also more favorable, but the deciding factor in management is the condition of the musculature as evidenced by the cardiac reserve force.

**Case III.**—Mrs. P., age forty-four, para XII.

*History.*—Patient is mentally unbalanced, has been in psychopathic institutions several times and is almost deaf. Patient makes the statement that she has had "heart trouble" (has been told so) for many years. She had no symptoms previous to delivery, at least complained of none; delivered six months ago, spontaneous labor, low forceps. There is no history of acute infections or beginning of heart trouble obtainable, so that the number of pregnancies she has gone through since the beginning of cardiac disease we do not know.

*Findings.*—Patient is a small, thin woman. Eyes react. Mouth and neck are negative. Arteries are hard; pulse is high and quick, regular, of the Corrigan type. Capillary pulse is moderate. Lungs are clear. Heart: Left border dulness is 15 cm. from midsternum (outside anterior axillary line), apex-beat is in fifth interspace and forcible. Right border at right edge of sternum. Systolic thrill is palpable in second right interspace at edge of sternum. Diastolic murmur of maximum intensity is heard at third left interspace and toward middle of sternum at same level. Systolic murmur maximum intensity is heard; second right interspace at edge of sternum, and is conducted along great vessels. Both murmurs are audible over whole precordium. Pulmonic second is accentuated. Aortic second is absent. Abdomen is negative. Reflexes are normal. Wassermann test is negative. Blood-pressure is 160/0. Diagnosis: Aortic insufficiency and aortitis.

**Case IV.**—Mrs. Z., No. 17,777, age twenty-three, para I.

*History.*—Patient complains of dyspnea on exertion, and edema which began about the third month of pregnancy.

*Previous History.*—There is nothing definite as regards heart except that dyspnea is always troublesome on exertion.

*Findings.*—When patient was first seen head and neck were negative. Arteries were soft, pulse was high and quick, Corrigan type, regular, capillary pulse. Lungs were clear. Heart: Left border cardiac dulness was 16 cm. from midsternum (midaxillary line), right border cardiac dulness was 3 cm. from midsternum. Apex-beat was diffuse, rather heaving, not strong, no thrills. There was a diastolic murmur at base, maximum intensity being in third left interspace at edge of sternum, rather small area. Pulmonic second sound was accentuated. Aortic second sound was absent. Cardiac tones were distant and rather weak. Blood-pressure was 120/40. Tones later improved in quality and double apical murmur became evident. Diagnosis: Aortic insufficiency, mitral insufficiency. Cesarean section and sterilization advised. Recovery good. Progress to date favorable.

What justified the difference in management of these two cases?

Case III, woman forty-four years of age, para XII. The age of child bearing probably soon past. She has a heart lesion of many years' duration, apparently the same lesion as at present, and with it has passed through repeated pregnancies and has no evidence of unfavorable progression.

We have a typical picture of aortic regurgitation, immense hypertrophy, Corrigan and capillary pulse, high systolic and pulse-pressure, diastolic murmur, and, in addition, an arteriosclerosis and aortitis, but the cardiac reserve is good. The hypertrophy has apparently kept pace with the demands made upon the heart and a state of compensation maintained, the heart has adapted itself to the life of the patient and has done so efficiently. We did not fear the possibility of failure from labor any more than from operation; operative procedure with her hypertension and vascular changes might be even more injurious.



Case IV, a young woman, para I, naturally wanted a living child, so pregnancy was carried to term. Her *primary complaint* is dyspnea, which is a warning of consumption of cardiac reserve force and the dyspnea is out of proportion to the mere addition of a pregnancy. The hypertrophy is so great that there is small possibility of much further adaptation of the musculature to increasing demands. She has weak heart tones, low blood-pressure, but otherwise a typical picture of aortic insufficiency. Physical findings not as extreme as in Case III, but the cardiac reserve force is not sufficient to adapt itself to the burden placed upon it. Even at rest there was some evidence of inefficiency and very moderate exertion provoked distress. Though there was no failure, a spontaneous labor might have been the last straw. We feared failure, which in these cases is sudden and without much warning. If an aortic insufficiency fails, that heart never returns to the degree of efficiency preceding failure.

Subsequent pregnancies would bring with them additional burdens and hazards. All of these were eliminated by cesarean section and sterilization, and though we cannot do away with all the burdens and dangers incident to the economic condition of the patient, we at least have less with which to contend in the maintenance of her present degree of health and cardiac efficiency.

Case III would be no better for having been operated, and the surgical risks would have been great.

Case IV suffered no injury by being operated, while definite possibilities of unhappy results, incident to spontaneous labor, were avoided.

#### SUMMARY

We feel that the management of cardiac complications of pregnancy should not be attempted on a basis of generalization of heart lesions, but that each patient should be individualized by observation by both obstetrician and internist and therapy instituted to suit each patient.

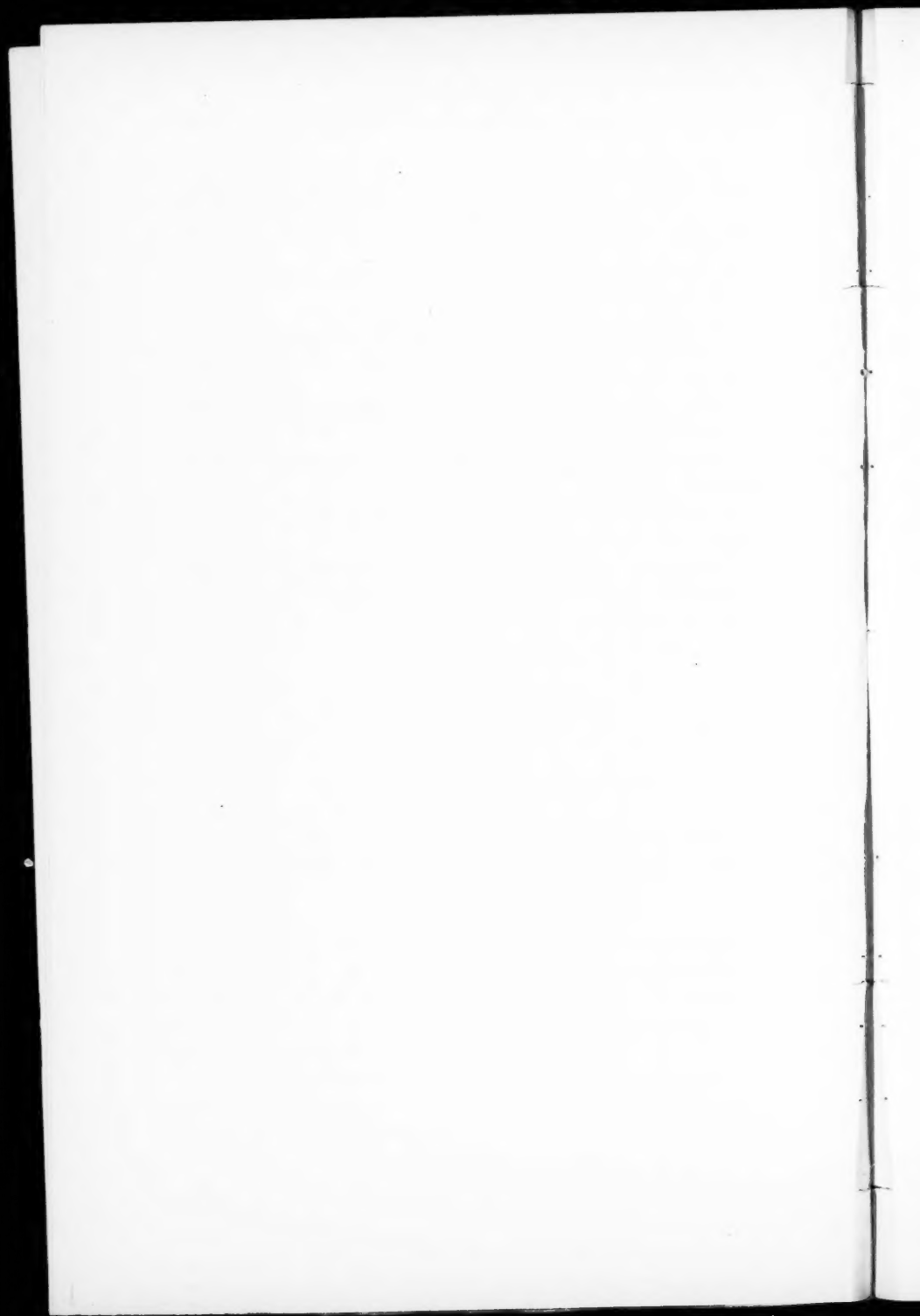
We do not believe that every patient requires operation, nor do we feel that every patient should be allowed to undergo spontaneous labor and delivery.



As a basis for individualization we have used a set of questions which we have tried to answer for each patient, regardless of anatomic lesion, and from the answers formed our decisions.

These questions inquired as to

1. Etiology and associated vascular or renal lesion.
2. Danger of actual failure.
3. Whether the damage is being increased.
4. Whether decreased voluntary activity will aid in maintaining cardiac ability.
5. Whether interruption or continuance of pregnancy will be more injurious.
6. Whether we can gain enough by abortion to warrant such procedure.
7. What are the signs by which we govern time of interruption?
8. Whether we should eliminate effort of parturition entirely or eliminate only the strain of the second stage?
9. Dangers of subsequent pregnancies.



## CLINIC OF DR. ROBERT SONNENSCHN

POST-GRADUATE MEDICAL SCHOOL

### ACUTE MASTOIDITIS AND SOME OF ITS COMPLICATIONS

**Unusual Prevalence of Infections of the Ear, Nose, Throat, and Air Passages in the Vicinity of Chicago During the Past Six or Eight Weeks. Acute Mastoiditis Frequently a Complication of These Infections. Anatomy of the Mastoid Process. Presentation of 5 Cases Illustrating Various Types of this Infection and its Complications.**

DURING the past six or eight weeks there has been a great prevalence of infections of the ear, nose, throat, and air passages in the vicinity of Chicago. These infections have caused many cases of acute otitis media which, in turn, has been followed in many instances by acute mastoiditis. It has seemed to some of us that a greater percentage of cases of otitis media was followed by acute mastoiditis than is usually the case. Another noteworthy fact is that so many of these mastoiditis cases after operation did not pursue the course that is noted in most instances. Whether this was due to a peculiar infection with *Streptococcus mucosus* (capsulatus), or whether other conditions were at fault, we are unable to explain. A number of the cases showed various complications, such as subperiosteal abscess, perisinus abscess, lateral sinus thrombosis, and pyemia. It is our good fortune to have a number of cases to demonstrate to you today which will illustrate some of the various phases of these complications. Before entering on this subject let us briefly consider the subject of acute mastoiditis, as such.

The mastoid process has been variously described as being of the pneumatic type, in which there are large air-cells; the sclerotic,

in which there are no cells at all, and the diplöetic, in which there is diplöe. At birth there is no real mastoid present, but there is a mastoid antrum which is a large cell communicating with the middle ear by means of the aditus ad antrum. The process of formation of air-cells or pneumatization proceeds from above downward toward the tip of the mastoid process. A fully developed mastoid process is not present before the end of the second year of life, but the pneumatization is not completed as a rule until the fifth or sixth year of life. In some instances we have, as Wittmaack pointed out, so-called arrest of pneumatization, the formation of the air-cells not taking place early, or sometimes not forming at all. However, in the normal mastoid process we can say that beginning with the antrum or the first mastoid cell communicating with the middle ear we have this process extending downward to the tip, so that in a great many cases we have the mastoid process filled with intercommunicating air spaces. It was shown by Politzer some years ago that in at least 95 per cent. of all cases of acute otitis media there is an extension of the inflammatory process from the middle ear by way of the aditus into the antrum and mastoid cells within the first few days of the onset of the trouble. It is this process which accounts for the fact that in so many instances we find pain in the mastoid process, tenderness on pressure upon the bone, and some fever during the first few days of the illness. The pain, tenderness, and temperature, however, usually disappear within the first week or ten days of the otitis media. The fact that the mastoid cells show an inflamed mucosa or even the presence of a secretion, mucoid or purulent, does not mean that the condition will proceed to a real mastoiditis with a breaking down of the walls between the little cells. It usually requires from two and one-half to three weeks before decalcification of the trabeculae takes place, so that the cells are merged into one another with formation of several cavities or one very large one. In the vast majority of the cases that we usually see the inflammatory condition in the mastoid cells subsides and does not recur, and the patient recovers from the otitis media. When, however, the bony partitions have broken down, and there is a profuse dis-

charge from the middle ear, so great that it cannot possibly come from so small a cavity as the *cavum tympani*, it is necessary to operate in order to clear out the broken-down cells and provide free drainage for the mastoid process and middle ear.

The indications for operation on cases of acute mastoiditis vary almost with every operator. The speaker a few years ago communicated with fifteen or twenty of the best known otologists in Europe regarding the subject, and also with a great many in this country, and found that the indications are different with each man. Some hold or think that acute suppuration from the middle ear, which in spite of treatment lasts for more than six or eight weeks, should be operated irrespective of the presence of any complication or not. Other men hold that unless serious complications of one kind or the other, intracranial or otherwise, appear, operation should not be performed merely because suppuration lasts a rather long period of time.

Therefore it is impossible to state categorically just what the indications for operation in acute mastoiditis should be. It may be said, however, that in the first week or ten days operation should only be performed if alarming symptoms arise, such as very high temperature which cannot be accounted for on any other grounds, such as pneumonia, etc.; second, marked swelling or very great tenderness of the mastoid process which does not respond to local applications of cold or heat, as the case may be; third, the presence of symptoms pointing to intracranial complication, such as very intense headache, stupor of the patient, delirium, etc.; fourth, the occurrence of symptoms pointing to involvement of the lateral sinus or the jugular bulb, such as chills followed by high temperature and remissions thereof. It is to be remembered that the jugular bulb lies just below the floor of the tympanic cavity, and in some instances the partition between these two is very thin or there may be a congenital dehiscence thereof. In that case it is possible to get a primary jugular bulb infection from the middle ear without any other symptoms, such as involvement of the mastoid process itself. In other words, if we have any very serious complications of any of the above-mentioned types, it is necessary to operate

early. If none of these symptoms are present and the patient only has moderate tenderness over the mastoid process, with only a moderate degree of temperature and with free drainage from the middle ear, it is best to wait for some time before interfering. However, if after two weeks the pain which was present in the first few days and then disappeared, recurs, or if the temperature recurs after having been normal, or if the tenderness or pain in the mastoid process returns, or if the amount of discharge increases in amount or continues very profuse from the beginning, coming in such great quantities that we can hardly clean the external auditory canal, the pus coming from the mastoid process and due no doubt to breaking down of the bony cells; when any or several of these conditions are noted two or three weeks after the onset of the trouble it may be necessary to operate. If there is no recurrence of pain or tenderness or temperature, if the discharge is unhindered from the middle ear, it may be advisable to wait three or four or more weeks after the onset, providing the patient has absolutely no other symptoms except free drainage. In young children sometimes after the suppuration has lasted six or eight weeks the whole condition subsides without operation, and the ear is apparently restored to its normal condition. You will see from this that it is impossible to state just exactly what the indications for interference are. It requires careful, complete examination of the patient, including blood findings, urinalysis, etc. One must be certain that leukocytosis or other changes are not due to some infection apart from the ear; in other words, a most careful inspection of all bodily functions must be made in order to arrive at a correct judgment regarding interference or non-interference. There are some conditions which present urgent indication for operation. These occur particularly in small children under the age of two or three in whom there is an open petrosquamosal suture permitting the escape of pus and the formation of a subperiosteal abscess. Where this occurs in infants or in older individuals prompt opening of the mastoid should be carried out. It is not sufficient merely to make the old Wilde incision, but it is necessary very carefully to examine the cortex, follow up the fistula when present, and with curet remove all

necrotic bone, completing the operation just as if no periosteal abscess had been present, but merely other indications which call for intervention. Another urgent indication for operation is the sudden appearance of paralysis of the facial nerve on the affected side. This is oftentimes due to the fact that the fallopian canal in which the facial nerve runs either has a congenital dehiscence, or the bone is eroded by the suppurative process and the nerve pressed upon. These cases should be operated upon very promptly, and when the pressure is removed the nerve slowly but, as a rule, definitely regains its function. I saw a case where on the tenth day of an acute otitis media paralysis of one side of the face occurred. Operation was performed on the same day. Within a week the nerve began to recover and within five or six weeks the face was almost normal.

We shall now have the pleasure of showing you a number of patients whom we have had under observation for some time one a simple, ordinary mastoiditis without any complications whatsoever, others with various findings in the mastoid process, such as osteitis, perisinus abscess, sinus thrombosis, etc.

**Case I.**—The first case we will present to you is a little girl, R. A., aged six, who came in some time ago with a history that she had had six weeks previously an acute tonsillitis during which time the left ear pained for several days, but no further signs developed. The left ear began to discharge intermittently four weeks ago. Appetite is good and she sleeps fairly well. During the past four days there has been terrible pain, swelling, and tenderness over the left mastoid process. Examination of the patient showed profuse discharge from the left ear. The drum membrane was not bulging and there was a fair sized opening in the lower posterior quadrant. The left mastoid process showed a moderate amount of redness, considerable tenderness on palpation, some infiltration of the skin and periosteum. The temperature was 99.5° F. per rectum and the condition of the patient quite good. Owing to the fact that there had been a four weeks' long suppuration and that there was now redness, swelling, and tenderness of the mastoid process, immediate operation was deemed necessary. She was operated upon the

next day under ether, and there was a great deal of infection of the soft tissues and breaking down of bone found. There was exposure of the lateral sinus and of the dura of the middle fossa, but no involvement of these structures. All necrotic bone was removed and a gauze packing inserted. The upper and lower ends of the incision were closed with silkworm-gut sutures and the main portion of the wound was packed. As is our usual custom, only the external dressings were changed the first two days. The main gauze packing was not removed until the fourth day, and then it was changed every day for about ten days, and after that every second day. There was an uneventful recovery, the child leaving the hospital on the tenth day after operation, and returning here for dressing. At the end of four and one-half weeks the wound had entirely closed and you see her today a well and happy little girl.

In this case there was a simple mastoiditis which, however, had already caused extension of the periosteum, so that there was some swelling externally; the bone was necrotic, with exposure of the lateral sinus and dura, but these tissues, as above mentioned, were not involved.

**Case II.**—This little patient, H. R., aged seventeen months, was brought in some time ago with the following history: Four weeks previously she had had a bronchitis which lasted seven to ten days. The baby recovered completely and for two days was entirely well. Then the temperature rose to 101° F. rectally and the patient continually picked at his right ear. The ear was examined, drum membranes were found injected, a diagnosis of double otitis media was made, and paracentesis performed. At first both ears drained very freely. The left ear stopped discharging in about a week and has been well ever since. In the right ear, however, the suppuration continued, and the temperature varied from normal to 101° F., usually being higher in the evening. Certain days the temperature would remain normal all the time, but on alternate days it would rise as before noted.

Examination of the patient now revealed nothing except the discharging right ear. As the perforation had become very small,



another paracentesis was done. There was cervical adenopathy on the right side, and considerable swelling of the posterior pharyngeal wall. Blood examination made the day of admission showed hemoglobin 65 per cent., red blood corpuscles 3,760,000, leukocytes 21,200, of which there were 60 per cent. neutrophils, 16 per cent. small mononuclears, 23 per cent. large mononuclears, and 1 per cent. eosinophils. The baby was kept in the hospital for twelve days, during which time the temperature varied from 98° F. per rectum to 104° F. Despite the continuance of the fever the parents took the patient home, where he remained for about seven days, apparently feeling better, but still ran an intermittent fever. On the seventh day after leaving the hospital there was a chill and the temperature rose to 104° F. per rectum. The child was then brought back to the hospital. At this time there was some swelling and tenderness over the right mastoid process.

When admitted for the second time the blood examination showed hemoglobin 60 per cent., red corpuscles 3,750,000, white corpuscles 26,100. The neutrophils were 58 per cent., the small mononuclears were 27 per cent., the large mononuclears 14 per cent., and 1 per cent. eosinophils. Examination of the urine both at this and the other time revealed nothing of a pathologic nature.

The following day the right mastoid was opened under ether. While there was no free pus in the mastoid cell, the entire process was filled with granulations. On removing these the lateral sinus was exposed, and while the wall looked a little bit rough it seemed otherwise intact and was not opened. The wound was then packed with gauze and the upper and lower ends of the incision closed with silkworm-gut suture. The patient's temperature then began to decline gradually, and he left the hospital two weeks after operation in apparently good condition. The wound, however, was rather slow in granulating and required about six weeks for closure. The baby is now well. It is interesting to note in this case that though there had been a rather free discharge from the ear during most of the time before he was operated, still at the time of opening the mastoid

very little free pus was found. The entire mastoid cavity, as above noted, was filled with granulations. Whether or not the presence of the latter in immediate contact with the lateral sinus had caused some inflammation or infection of the wall of this large venous channel we cannot say. It is likely, however, that this was the cause of the chill which occurred the day before the operation. While one cannot prove the assertion, still it seems fair to assume that had the child not been operated upon for another four or five days, a marked involvement of the sinus wall might have taken place, with the possibility of a sinus thrombosis and pyemia.

**Case III.**—Our next patient, B. S., aged fifty, office worker, has a rather long and complicated history. He first entered the hospital some months ago, complaining of severe headaches, tenderness over the left mastoid region, dizziness, forgetfulness, vomiting, impaired hearing, and at times chills. Four weeks prior to entrance into the hospital the patient began to experience pain on the left side of the forehead and soreness in the left mastoid region for two or three days. He also complained of severe pain in this region with vertigo. There has been impairment of hearing in the right ear for seven or eight years. Two years ago this ear suppurated for a short time and again eight months ago, but not since then. A few months ago the patient had had an infection in his left ear, and at one time it was thought necessary to operate the mastoid on that side, but the suppuration subsided without operation. The hearing, however, on the left side has also become considerably impaired. For the last two days prior to entrance into the hospital the patient was drowsy and it was difficult to arouse him. During those days he also complained of loss of memory, vomiting, which was projectile in type, and chills. There is nothing in the previous history of the patient to throw any light upon the present complaint or, rather, the diagnosis thereof.

Examination of the patient's nose showed a deviation of the septum to the right and a large polyp in the right middle meatus. The tonsils were submerged and contained caseous material in crypts. Examination of the ears showed a retracted drum mem-

brane on the left side and an injected one on the right side and a small amount of purulent discharge. With pressure upon the mastoid processes he would complain of tenderness first on one side and then on the other, and this reaction also varied on different days, one side being more sensitive than the other, so it is very difficult to say which was the more tender. Examination by the neurologist showed that there was considerable ataxia. Pupils were regular and equal on both sides. Ankle-jerk was increased, but there was no clonus. There was a suggestion of a right Babinski, but no Chadwick reflex. On the right side there was a positive Gordon and Oppenheim. The same findings were had on the left side as on the right. Walking was rather difficult for the patient and he swayed toward the right. Examination of the eyes by the ophthalmologist showed the right fundus disk edges slightly blurred and more pink than normal. Physiologic excavation elevated less than 1 diopter; no exudate was present. Arteries smaller than normal and somewhat constricted. Arteries not more than one-third the normal size. All vessels tortuous, especially the smaller branches. Retina distinctly hyperemic; no hemorrhages or exudate present. The left fundus was practically the same as the right, except the left disk was slightly elevated and the edges slightly blurred. The diagnosis made was bilateral retinal sclerosis with incipient choked disk. Later re-examinations of the eyes showed no change in these findings.

Complete vestibular tests were then made. There was no spontaneous nystagmus to either side. There was no spontaneous past-pointing with the right hand, but with the left hand there was slight past pointing toward the right. Turning to the right gave a horizontal nystagmus of twenty-five seconds and turning to the left gave a nystagmus of twenty-five seconds. With vertigo tests there was very marked reaction, especially on rotation to the left. The caloric tests with water at 68° F. showed no reaction of the left ear after douching for six minutes. When the head was bent backward, however, the horizontal canals reacted strongly. Then bringing the head to the erect position the vertical canal reacted well with rotatory nystagmus to the right and past-pointing to the left. Douching right ear for five

minutes gave no response of the vertical canals. With the head thrown back the horizontal canals reacted markedly. Elevation of the head then showed vertical nystagmus to the left with past-pointing to the right. The caloric test showed overaction of the right horizontal canals, but delayed action of the right vertical canals. On the left side there was delayed action of both the vertical and horizontal canals. Falling reaction—five rapid turns to the right with the head falling to the right; five rapid turns to the left with the head falling to the left. For whispered voice there was no hearing at all in the right ear, but conversation was heard at a distance of 1 foot. On the left side whisper was heard at a distance of 4 inches. In both ears the lowest and the highest octaves were not heard by means of tuning-forks.

Following expectant treatment the patient gradually improved and the headaches disappeared. He was then sent home and told to report if any further symptoms developed. Let me say that throughout his stay in the hospital repeated examinations of the urine were negative. Blood-count showed hemoglobin 80 per cent.; red cells 4,200,000, and the white blood-corpuscles varied from 11,000 to 14,600. Blood-pressure was systolic 115 and diastolic 85. Blood-cultures were negative and likewise the blood Wassermann. Spinal puncture showed a cell count of 11, Lange, Ross-Jones culture, and Wassermann were all negative.  $\alpha$ -Ray examination of the skull was negative except that the cortex of the right mastoid seemed more dense than that of the left. Temperature, pulse, and respirations were normal throughout. It is difficult to say what caused the symptoms of cerebellar ataxia together with other vestibular findings. A few days after leaving the hospital the patient began to notice pain and tenderness in the right mastoid region. He was then readmitted, and we found some tenderness and swelling just behind the tip of the right mastoid process. Examination by the neurologist showed no paresis. The motor cranial nerves were normal, the pupils reacted both to light and accommodation normally, cerebellar functions were normal, there was no adiadochokinesis. There was no hypertonia. Ophthalmoscopic examination showed both right and left disks practically normal. Veins of

the right eye did not dilate or pulsate on extreme pressure over right jugular vein, but there is a distinct darkening of the veins of the fundus. The test could not be called positive, but merely suspicious. Because of the tenderness and swelling at the tip of the right mastoid process operation was performed.

It was not until the lateral sinus was exposed that a great deal of pus was found. This bathed the sinus and it was necessary to explore far posteriorly before an area could be found that was free from pus. The cortex of the mastoid and occipital bone was found to be necrotic for a considerable area, especially extending posteriorly over the lateral sinus. Culture of this pus showed pneumococcus. In view of the fact that there was no sign of cerebellar involvement the brain was not explored. The very large wound was packed with gauze and the patient made an uneventful recovery.

Here we have a very puzzling case, first with the history of cerebellar ataxia and finally with external symptoms, such as swelling of the right mastoid. At operation we found an immense perisinus abscess with bony necrosis over a large area of the external cortex of the mastoid and occiput. Whether this pus had been present for some time, say several weeks, and had caused pressure upon the cerebellum, which pressure was later relieved when the pus was able to burrow externally or not, we cannot say. It is interesting to note, however, that at the time of operation the sinus was found bathed in pus, but no thrombosis thereof, and that on simply removing the necrotic bone and proceeding as we do in an ordinary mastoid operation, the patient was entirely relieved and is now well, as you can see for yourself. Had more definite signs of localization in the cerebellum been present when the patient was in the hospital the first time we would have operated, but as the case later proved we would have found nothing there, the entire condition being in the mastoid process. It is in cases like this, obscure at first, but carefully studied for several weeks, that so much information may finally be obtained.

**Case IV.**—Our next case, D. S., aged eight, is the little girl who stands before you. The history is long and interesting.

This young lady was quite well until six months ago when, following a head cold, she developed pain in the left ear. This was followed by suppuration lasting four weeks, at the end of which time a competent otologist operated because of mastoiditis. The patient got along nicely and left the hospital ten days later, returning daily for dressing. About two weeks after the operation she began to have some fever. She had no chills and there was no sudden rise in the temperature, but it would climb up to about 101° F. On the fifth day there was a gradual rise in the temperature from 99° F. in the morning to 105° F. at 6 o'clock in the evening, without a chill. It was at this time that we saw her, and while it was impossible to say what was causing the temperature, we kept her under observation for a few days after her return. The blood-count on the day of readmission to the clinic was 22,000 white blood-corpuscles. The polymorphonuclears were 74 per cent., lymphocytes 22 per cent., and large mononuclears 4 per cent. The urine was entirely negative as regards to sugar, albumin, casts, etc. During the next few days the white blood-corpuscles varied in number from 15,000 to 22,000. During this time the temperature varied from 101° to 105° F. per rectum. She had no definite rigor, but at times felt chilly.

On the sixth day after readmission to the clinic the patient was again operated. The old wound was entirely reopened and a great many granulations removed. The lateral sinus was exposed, and although it seemed a little rough, it was free from thrombus and was not opened at the time of the operation. During the next four days the patient's condition seemed somewhat improved. Temperature was considerably lower and she was quite cheerful. On the fourth day she had a chill and the temperature rose again to 105° F. The wound was quickly reopened, the lateral sinus found to be thrombosed, and the thrombus removed until there was free bleeding from both ends of the sinus and from the jugular bulb. Owing to the free bleeding which was obtained from the lower end of the sinus it did not seem necessary to ligate the jugular vein. The wound was then repacked and the patient returned to bed. After this last opera-

tion the count of white cells began to diminish and got as low as 7800. The patient seemed a great deal better, the temperature for several days at a time remaining as low as 99° F. At other times it would rise to 100° or 103° F. The patient, however, developed a pallor and looked more toxic, and it was deemed advisable to hold consultation. At this time it was decided to tie off the jugular vein, but the parents refused to permit any further intervention. As before stated, at the last operation bleeding was free from both ends of the sinus, and therefore we did not ligate the jugular, but because of the symptoms that occurred afterward we regretted not having done so. There evidently was a small obturating or possibly only a mural thrombus extending to or beyond the jugular bulb which was causing the pyemia now present. Because of the parents' decision we could do nothing more than use supportive measures and keep the patient absolutely quiet. The mastoid wound was healing very nicely, and finally the patient went home. During the first week her temperature stayed very low, but suddenly she had two very severe chills and temperature rose to 106° F. Despite this untoward symptom, however, she gradually improved, and finally entirely recovered after an illness of four months. You can see that she is now, two months later, a fat, chubby little girl, apparently in perfect health despite her long and serious illness.

Here, then, we have a case of sinus thrombosis which apparently occurred after the second mastoid operation, due possibly to the fact that the jugular vein was not ligated. The patient finally recovered, showing what a wonderful resistance some individuals have to very serious infection. We are happy to know that although this patient had embolism no definite foci of infection appeared in the joints or elsewhere, such as one might expect. It is impossible, of course, to state how much this course might have been shortened had we at first ligated the jugular or had later been permitted by the parents to do so. We are most fortunate to have had a recovery in a case which showed definite symptoms of pyemia.

**Case V.**—Our fifth and last case is this little girl, D. L., aged



ten. One week before admission to the hospital the patient was brought to us with the history that for three weeks previous there had been some discharge from the left ear, with marked tenderness over the left mastoid process during the last twenty-four hours. Examination of the left ear showed a reddened drum membrane with a small central perforation. Under gas anesthesia a very wide paracentesis was performed. There was very free drainage and within twenty-four hours the tenderness over the mastoid had entirely disappeared. During the next four or five days the patient complained of considerable frontal headache and there was a rise in temperature to 100° F., and sometimes to 101° F. Examination of the nose showed no signs of sinusitis which might account for the frontal headache. It was then decided that there was probably some irritation in the posterior cerebral fossa which accounted for the peculiar headaches, as is frequently the case. She also had vertigo, but absolutely no signs of nystagmus or of vestibular irritation. At the time of her admission to the hospital the eyes were examined ophthalmoscopically and the fundi showed dilated retinal vessels, curving over the margins of an edematous disk, no hemorrhages, or retinal or choroidal lesions. The height of the summit of the disk over the retina was 1½ mm. The diagnosis was that of bilateral choked disk. x-Ray examination showed the right mastoid clear, but the left one rather hazy, with a breaking down of the trabeculae. The day after admission to the hospital the left mastoid was opened. The lateral sinus was found lying far forward directly over the mastoid antrum and was seen to be entirely thrombosed. The dura covering the posterior cerebral fossa was explored, but no lesion found. The lateral sinus was then opened and a very firm thrombus removed. Free bleeding was had from both ends, and therefore the jugular was not tied. Blood-counts made on the day of admission showed 26,000 leukocytes and on the day of operation 19,000, with polymorphonuclear neutrophils 77 per cent., small mononuclears 16 per cent., large mononuclears 7 per cent.

For the first three days after this operation the patient did very well and the temperature remained low, but on the fourth



day there was a marked chill, the first one she had had since the onset of her trouble, followed by a rapid rise in temperature to 105.6° F. On this day the leukocyte count was 18,600. We then at once exposed the left jugular vein and ligated it. Her condition on the table was very poor, and therefore no effort was made to tie off separately the facial and other veins leading to the jugular. The mastoid wound was reopened, cleaned, and packed. From this time on the patient made an uneventful recovery. On the twelfth day after ligation of the jugular she left the hospital. The mastoid healed promptly, and as you see the patient today she is in perfect health, and is considerably heavier than before the operation. Repeated examinations of the eye-grounds have shown that the choked disk is gradually but surely disappearing.

Here we have a case that presents a history somewhat similar to the preceding one except that the course was much shorter, and here also, because there was free bleeding from the jugular bulb, ligation was not performed. Four days later severe chill occurred and then the vein was tied. Some operators contend that whenever you find a thrombosis of the lateral sinus it is best to ligate the internal jugular vein at once because it is impossible in many instances to remove all the clot, and as a result a new thrombus of the jugular bulb or below that point may cause further embolism. That may be true, but we know from our own experience that in quite a number of other cases a mere removal of a thrombus was followed by complete recovery without tying the jugular, so it is still an open question just when to do a ligation. Sometimes when we have a pyemia we expose and open the sinus, do not find a thrombosis, and yet there may be an obturating or a mural thrombus farther down which continues the pyemia. In individual cases it is necessary to weigh all the evidence and to use one's judgment based on his own experience and that of other otologists.

With reference to the diagnosis of the presence of lateral sinus thrombosis it is well to remember that if, for instance, the right sinus were blocked, pressure upon the opposite internal jugular vein would cause passive congestion of the retinal veins. But

this sign is not always a positive indication nor is it always present.

We have this afternoon shown you 5 cases; the first one was a simple mastoiditis with the typical findings at the time of operation. In the second case we had an acute mastoiditis without any great accumulation of pus, but the entire mastoid filled with granulations. In the third case there was a very large perisinus abscess without sinus thrombosis, but with a great many symptoms of cerebellar involvement. You will recall, however, that before operation all symptoms of cerebral ataxia had disappeared. When we did open the mastoid we found a great area of necrotic bone extending posteriorly. The next case was operated several times. Finally, a sinus thrombosis was found, the clot was removed, but the pyemia continued, and although jugular ligation was advised, the operation was refused. The last case was also one of pyemia. We waited four days after the primary operation, then a chill occurred, and the jugular vein ligated. All of these cases made good recoveries and we were most fortunate in this regard. The history of these cases will show you how many complications may occur in the course of an otitis media, especially an acute mastoiditis. It is therefore absolutely necessary if you have a case of middle-ear suppuration to watch it most carefully. Early paracentesis is very often a good preventive of further trouble, although it is not certain. However timely, it may not avert a mastoiditis. As I said at the beginning of the hour, in the vast majority of cases of otitis media there is some inflammatory reaction in the mastoid cells during the first week of the infection. However, it is only in a small percentage of the cases that the bony trabeculae break down and actual mastoiditis occurs. Careful examination, early paracentesis, blood examination, x-ray, and other findings are necessary before one can decide when to operate and when not. Any mastoiditis, however simple it at first appears, may have serious complications which not infrequently prove fatal.

## CLINIC OF DR. JULIUS H. HESS

COOK COUNTY HOSPITAL

### FRIEDREICH'S ATAXIA IN TWIN BOYS

**Twin Boys Showing Progressive Changes in Their Nervous Systems Referable to Spinal Cord, Cerebellum, and Probably Cerebrum Following an Attack of Epidemic Influenza in 1918. Etiology, Pathology, and Diagnosis. Difficulties Encountered in the Diagnosis of Such Cases.**

THE cases which we are to present this morning are of especial interest because of the almost simultaneous occurrence and the similarity of onset and course of the nervous system lesions in these twin boys.

**Case I.**—T. H., white schoolboy, aged ten, admitted to Cook County Hospital May 17, 1920, with the following history: All of the children in the family, numbering 7, were stricken with influenza in October, 1918. During convalescence 5 of the children quickly recovered, but the patient and his twin brother remained unusually weak and were not able to eat, dress themselves, or walk because of ataxic extremities. Their heads wobbled on their shoulders. Gradually, in the course of a few months, they partially regained control of their movements sufficiently to walk about, dress themselves, and eat, but all with difficulty. At the time of their first admission to the hospital, over two years after the onset, the mother stated that the patients made aimless, involuntary movements of the head, limbs, and trunk, made silly appearing involuntary grimaces, dropped things easily, had an awkward, reeling, staggering gait, and general muscular weakness evidenced by drooping shoulders and generalized relaxation of the trunk muscles. The condition was gradually getting worse with temporary remissions. Both

boys were doing very well in school until their illness in 1918, since which time Thomas had been making slow progress, while his brother showed a mental age less than that of his preataxic days.

*Previous History.*—Patient was one of twins, born at the ninth month and breast fed for six months. He was always a healthy normal child until the attack of "flu" except for measles when quite young. Tonsillectomy was performed four years ago. He frequently boxed skilfully with his twin brother for exhibitions. This is emphasized to show the state of development of their nervous and muscular systems before the illness.

*Family History.*—Father and mother were living and well. There were 5 other children in the family, living and well. No children had died and there had been no miscarriages. The twin brother had symptoms similar to those presented by the patient. Relatives all had large families of healthy children. *As far as known there was no similar disturbance in other members of the family.*

*Physical Examination.*—The patient was a fairly well-nourished white boy of ten years, evidently not acutely ill. He stood in a stooping attitude, with round shoulders, and the trunk held forward. There were irregular oscillatory movements of the head and trunk.

*Head:* Face was masklike in appearance and expressionless. Pupils were equal and regular and reacted to light and accommodation. There was no mystagmus or optic atrophy or other eye-ground changes. Vision was below normal. The teeth present were in good condition. There was a gross tremor of the tongue. Tonsils had been removed.

*Chest:* The chest was asymmetric. There was a dorsolumbar scoliosis of marked degree. Lungs were negative. Heart rate was normal. The apex was 1 cm. outside of the midclavicular line. There was a soft blowing systolic murmur at the apex.

Muscular development was poor.

*Extremities:* There was a pes cavus present. There was no tremor of arms or hands.

*Co-ordination:* Patient was able to maintain handclasp and to

keep the tongue protruded. The gait was awkward and exaggerated. The feet were widely separated in standing and there was a tendency to stumble. The patient could pin up his gown with a fair degree of success. Quick turning or reversing his direction caused excessive movements of the arms and upper trunk to maintain his balance. At times he kept from falling by grasping stationary objects.

Tactile and muscle senses were intact. Pain and temperature senses were normal.

Muscle strength was far below normal. This point is interesting in view of his having been very adept at boxing.

Bladder and rectal control were good.

The speech was distinctly slowed, with a moderate degree of scanning. His memory was poor as to names, places, and dates.

Reflexes: The Romberg sign was present to a marked degree. The pupillary reflexes were present, but sluggish. The upper extremity reflexes were intact. The knee and Achilles' jerks were absent. A bilateral Babinski was present.

*Laboratory Findings.*—The urine was negative. The leucocyte count was 7200 and the blood Wassermann was negative.

*Subsequent History.*—The patient returned to the hospital June 2, 1921. The mother stated that the swaggering gait had become more pronounced and the muscular weakness and clumsiness more prominent. He had not re-entered school because the mother did not think he was capable of doing the work. Some changes were noted in his physical condition. An alternating horizontal nystagmus had developed and was present when the patient looked at an object to the left of the median line. The quick component of the nystagmus was to the left, the slow to the right and the opposite was true when he looked at an object to the right of the median line.

The arches of the feet were very high. The big toe approached the "hammer-toe" type, with the first phalanx in extension.

The gait was that of a drunken man, swinging his arms in an attempt to balance himself while endeavoring to walk in a straight line and turning wide corners. The gait was staggering and at times he would lose his balance and fall over.

The changes noted in the reflexes were that the biceps and triceps and wrist reflexes were absent. The Oppenheim sign was present, but the Gordon was not. The Babinski on the right side was more marked. The abdominal and cremasteric reflexes were present.

Speech was considerably slowed and scanning in character.

The patient returned to the hospital for a third observation in February, 1922. At this time the Romberg was somewhat less marked, as was also muscle sensation. The reflexes were the same as on the previous examination. The nystagmus had practically disappeared except upon prolonged exertion. Because of his inability to concentrate and otherwise apply himself he had been refused entrance to the public schools. Memory tests showed little alteration from those of eight months previous.

**Case II.**—S. H., twin brother of Thomas, age ten years, was admitted to the Cook County Hospital on the service of Dr. W. F. Winholt May 17, 1920, with practically the same history as that of his twin brother.

*Physical examination* revealed approximately the same findings as that of the brother, but present to a lesser degree. His mentality was better and his co-ordination somewhat more accurate. The more important findings were the following:

**Head:** Pupils were equal and regular and reacted fairly well to light and good to accommodation. Jerking nystagmus was present at times. There was no marked tremor of the tongue, yet it was somewhat unsteady.

The spine showed the presence of a slight scoliosis.

**Reflexes:** The biceps was present on both sides. The kneejerks were absent. The Oppenheim and Babinski signs were present and the Romberg was marked.

Muscular development was poor, but better than that of his brother.

He walked with an ataxic gait and a tendency to use the ball of the foot. He was able to get about alone. Pes cavus was present on both sides.

*Laboratory Findings.*—Wassermann test on the blood was negative.

*Subsequent History.*—On October 22, 1920 the patient returned to the hospital suffering from arsenical poisoning which developed while taking Fowler's solution. There was no change in his physical condition.

On June 2, 1921 he returned to the hospital for a third observation. At this time there was an alternating nystagmus. When he looked at an object to the left of the median line the quick component was to the left, and vice versa when he looked at an object to the right of the median line. The gait was staggering and simulated that of a drunken man. He swayed his arms to help him keep balanced and was unable to walk in a straight line. Speech was scanning.

Reflexes: The biceps, triceps, and wrist reflexes were absent. The Babinski and Oppenheim were more marked and the Gordon was present. The cremasteric and abdominal reflexes were present.

On the whole, the condition had been progressive.

In February, 1922 he again reported at the hospital. The changes noted in his condition were slight except for the fact that his self-confidence and mental activity were improved.

#### DISCUSSION

**Etiology.**—There has been some doubt in our minds as to the exact classification of these cases because of the absence of hereditary history. However, the familial history, in that the disease is present in these two members of the family, may be considered as supporting evidence for Friedreich's ataxia. The early age of the development and the fact that the lesions followed an acute infectious disease are corroborative factors and correspond with most of the reported cases. It is also of interest to note that of the cases reported there has been a preponderance in males, while the transmission has usually been by the female line in cases where a hereditary history is present. There is no history of lues, alcoholism, insanity, or neuropathic tendency in the family. The interesting problem which goes unanswered in these boys is the question as to whether the acute infection resulted in degenerations in congenitally defective nervous



systems, or whether we were dealing with boys with a normal mental and physical development who have undergone an acute infection of the nervous system resulting in degeneration and sclerosis.

**Pathology.**—Based upon the general manifestations of degeneration of the nervous system, of which the most marked lesions are referable to the spinal cord, with mental changes and ocular manifestations, which indicate involvement to a lesser degree of the cerebrum and cerebellum, we are unquestionably dealing with a generalized involvement of the nervous system. The reported autopsies from various sources agree quite closely at least on the location of the cord changes, although, as might be expected, the degree of involvement of the various tracts varies greatly in individual reports. The tracts most commonly involved are the columns of Goll and Burdach, the crossed pyramidal tract, Gower's tract, direct cerebellar tract, and in some instances Lissauer's tract and Clarke's column. The cerebral and cerebellar regions described have differed more widely in their distribution and character, the latter being more commonly involved, while many of the cases show no early mental disturbances.

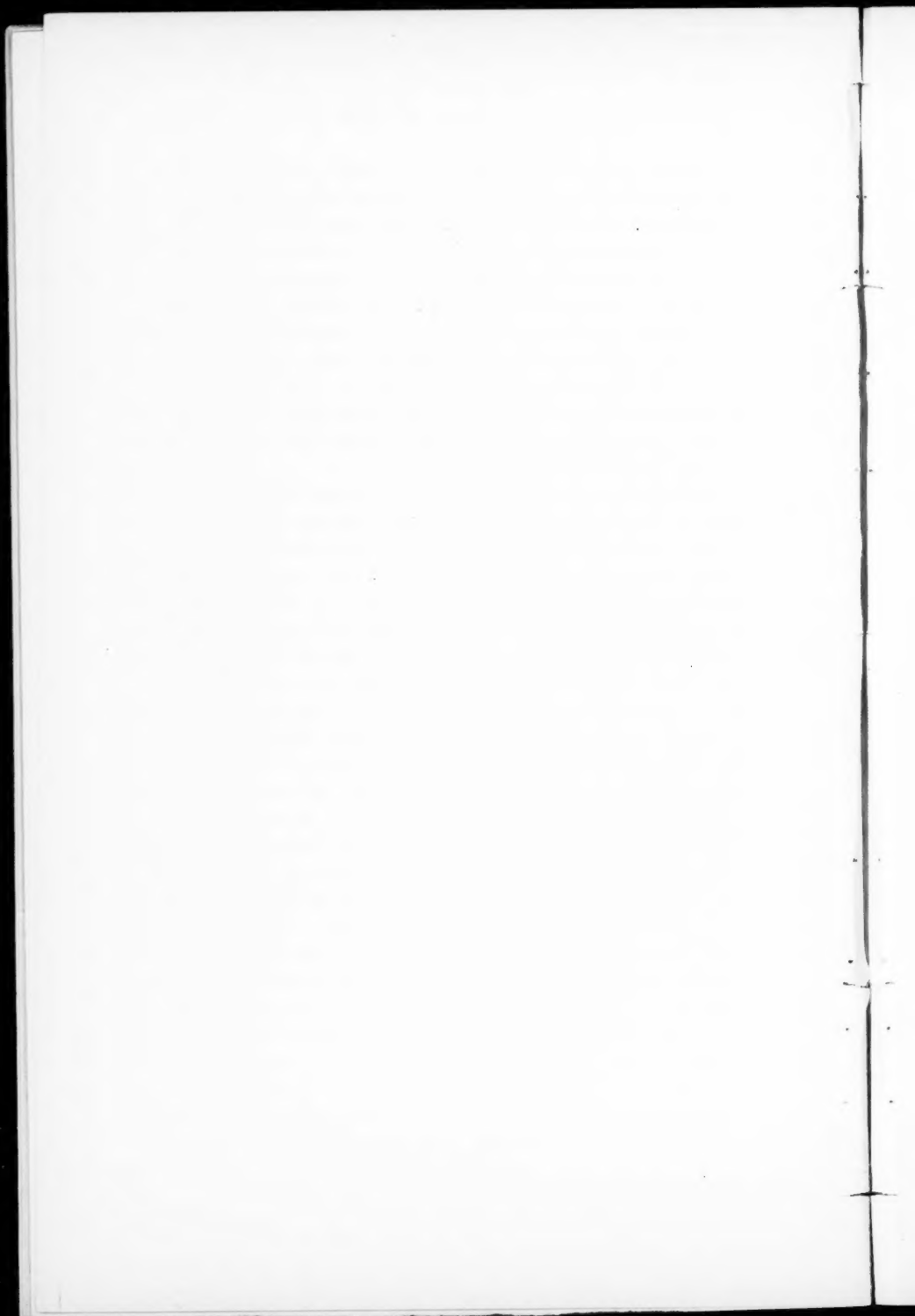
**Diagnosis.**—In boys of this age several conditions must be differentiated. The acute forms of encephalitis and myelitis of infectious origin have already been mentioned. *Marie's form* of hereditary cerebellar ataxia can be excluded on the ground of its late development, the common presence of optic atrophy, spasticity of extremities associated with increased reflexes and foot clonus, and the usual freedom from deformities of the foot and spine. In the *juvenile form of tabes* there is an absence of a cerebellar quality to the gait, while disturbances in the pupils, sensation, visceral crises, bladder, and rectal symptoms develop at some stage of the disease together with the confirming findings of stigmata of syphilis and positive Wassermann reaction. *Multiple sclerosis* resembles Friedreich's ataxia in that several members of the same family may be affected. It is often most difficult to diagnose from Marie's form when it occurs in early adult life. It may be excluded in these young boys, however, on



the ground that it is accompanied by spastic ataxia, together with increased reflexes, intention tremor, and optic changes.

**Summary.**—We find twin boys aged ten years who first showed progressive changes in their nervous systems, referable to the spinal cord, cerebellum, and probably cerebrum at the age of eight years. The boys were of an athletic type, being expert boxers and well advanced in their school work before they became ill with influenza. Following this acute infection they had a very slow convalescence, during which their nervous manifestations became apparent. These have been progressive in both boys, one, however, showing somewhat less involvement than the other.

The family history is negative upon careful inquiry. The onset in these cases following an acute infection is typical of many of the recorded cases. In view of the increasing nervous system lesions following acute infections, such as encephalitis, involving all sections of the brain with their varied clinical pictures, as well as the many atypical varieties of meningitis and spinal cord lesions more recently reported, the question naturally arises in the cases of the twins, in the absence of a family history, as to whether the boys are cases of true hereditary ataxias of the Friedrich types or cases of acute combined posterior lateral sclerosis with simultaneous cerebellar and cerebral involvement secondary to the infection which preceded the onset of the nervous manifestations.



## CLINIC OF DR. FRED M. SMITH

PRESBYTERIAN HOSPITAL

### THE TREATMENT OF PREMATURE CARDIAC CONTRACTIONS WITH QUINIDIN SULPHATE

**Presentation of 5 Patients with Premature Contractions. Use of Quinidin in this Condition. Effects of this Drug as Viewed by Other Authors.**

THE frequent appearance of premature contractions even in the absence of other cardiac symptoms is often very alarming to the patient. The drugs that are ordinarily employed in the treatment of other cardiac conditions apparently do not influence the prevalence of this disorder.

In 1918 Frey<sup>1</sup> discovered that quinidin, the dextrorotary stereo-isomer of quinin, would restore the sinus rhythm in some instances of auricular fibrillation. Since then more than 300 patients with absolute irregularity have been treated abroad and in this country. In about 50 per cent. a regular cardiac action was established. The value of quinidin in the treatment of other types of irregularities has received very little attention. Boden and Neukirk<sup>2</sup> apparently had favorable results in the treatment of simple paroxysmal tachycardia and premature contractions with this remedy. White, Marvin, and Burwell<sup>3</sup> have recently mentioned one instance in which ventricular premature contractions disappear following the administration of quinidin sulphate. In 20 patients<sup>4</sup> the premature beats were eliminated in 10 and the frequency diminished in 7 by quinidin. The following patients have been under observation for periods of three to five months. In each there has been sufficient time to determine the value of the quinidin in the treatment of the

premature contractions. These results are fairly illustrative of our experience with this method of treatment.

**Case I.**—E. O., age forty-four, janitor, entered hospital November 10, 1921, complaining of an irregular action of the heart which had occurred almost daily for the last five months. He stated that he was weak, nervous, and had difficulty in sleeping. The appetite was poor and he had lost 15 pounds in weight. He had been unable to work for two weeks because of weakness. He had been treated during his early life for pulmonary tuberculosis. His health had otherwise been good. He denied venereal disease and there was no history of mis-carriages.

The physical examination was negative except for the appearance of a premature contraction after every fifth to tenth normal contraction. The heart was apparently normal in size and there were no murmurs. The systolic blood-pressure was 110 and the diastolic was 80. The urine was negative. There was a moderate secondary anemia. The Wassermann test on the blood was negative. The electrocardiogram showed left ventricular premature contractions. There were no other electrocardiographic evidences of cardiac disease.

This patient was given quinidin sulphate 3 grains after meals. After the first dose the heart became regular. He stated that this was the first time that his heart had remained regular for more than a few hours for weeks. He was discharged from the hospital on the tenth day. During this time there had been no recurrence of the irregularity. He returned to the cardiac clinic two weeks later and reported that the heart was still regular. He had gained 8 pounds in weight, was sleeping well, and was feeling very much better generally. The patient has since been seen at two and three week intervals. The quinidin has been reduced to one dose a day. He recently reported that he had gained 18 pounds in weight and was feeling stronger than he had in months. An occasional skip was felt in the radial pulse, but the patient was apparently unconscious of it.

**Case II.**—J. S., age sixty-four, porter, came to the cardiac

clinic November 30, 1921 for attacks of dizziness. These attacks had appeared at irregular intervals for the last four years. During the last six months they had occurred daily. At the time of the dizziness the heart seemed to stop and there was a sensation of gone-ness in the precordium that extended to the throat. The patient was not short of breath and there was no history of chest pain. He stated that he contracted syphilis at the age of twenty-two and was treated for a gumma of the sternum at forty-four. Since then he has had repeated courses of mercury and iodids and several salvarsans. The Wassermann test on the blood was reported negative. He has 3 children and there have been no miscarriages.

He was examined carefully for signs of syphilis, and none were found except for the scar on the sternum and a few copper-colored scars on the shins. The heart was apparently normal in size. A faint systolic murmur was heard over the aortic area and there was an occasional premature contraction. The systolic blood-pressure was 135 and the diastolic 80. The urine was negative. The electrocardiogram showed a slight left ventricular preponderance and left ventricular premature contractions.

The patient was instructed to take a capsule containing 3 grains of quinidin sulphate after meals. He reported in one week that he had been free of the dizziness and the irregularity. On December 14th the quinidin was reduced to two doses a day, and on January 22d was further reduced to one dose a day. During this time he had not been disturbed with skipped beats, was sleeping well, and felt much better generally. On February 18th the quinidin was discontinued. In about ten days the patient reported that the trouble had returned. He was again put on the quinidin once a day and is now getting along very well.

**Case III.**—Mrs. P. F., age thirty-four, came to the cardiac clinic December 17, 1921. She complained of precordial pain which was accompanied by a jumping of the heart. This trouble had appeared at irregular intervals during the last two years, but had been very frequent the last few months. She was

nervous, unable to sleep, and felt weak. She had been compelled to stop her work because of general weakness. She had had one miscarriage. The past history was otherwise negative.

The physical examination was negative except for an occasional premature contraction. The systolic blood-pressure was 125 and the diastolic 75. The Wassermann test of the blood was negative. There were no evidences of kidney trouble. The electrocardiogram showed right ventricular premature contractions.

The patient was prescribed capsules containing 3 grains of quinidin sulphate, and instructed to take one capsule after each meal. The premature beats were eliminated. On January 18th the medication was reduced to one dose about one hour before bedtime. She had told us that the premature contractions were more frequent soon after retiring for the night. She reported February 1st that her heart was still regular. Her general condition was apparently much improved. The quinidin was discontinued at this time for one week. The skipped beats returned. She returned to the one capsule one hour before bedtime and has practically regained her normal health.

**Case IV.**—Mrs. F., age thirty-five, telephone operator, complained of palpitation of the heart and precordial pain. This trouble had been present for about six months. At first it was noticed at infrequent intervals. During the last month it has appeared almost daily and frequently many times during the day. The past history was negative.

The physical examination was negative except for an occasional premature contraction. During the examination it was noted that the patient was conscious of every skipped beat. This was apparently responsible for the palpitation and precordial distress. There were no abnormalities in the electrocardiogram. No premature contractions were caught. The blood Wassermann was negative. There were no evidences of kidney disease.

The patient was prescribed quinidin sulphate 3 grains after meals. She was encouraged and instructed in regard to her

diet and general habits of living. She reported in one week that her cardiac distress disappeared after beginning the quinidin. She later returned at irregular intervals and at times was out of medicine. During the time that she took the medicine she was unconscious of any irregular action of the heart. As soon, however, as she discontinued the quinidin the trouble returned. Recently, however, the medication apparently does not influence the prevalence of the premature beats. The patient is becoming extremely nervous and is greatly concerned over her condition.

**Case V.**—Mrs. G., age thirty-eight, entered the hospital July 15, 1921. She told us that she had had an irregular action of the heart for four years. She was of an extremely nervous temperament and had frequent severe attacks of migraine. There was apparently nothing significant in the past history.

The heart was normal in size. There were no murmurs. Frequent premature contractions were noted on numerous occasions. The electrocardiogram showed these to be left ventricular in type. The systolic blood-pressure was 125 and the diastolic 85. The Wassermann test on the blood was reported negative. There were no pathologic findings in the urine.

This patient was given quinidin sulphate 3 grains after meals. The dose was later increased to 6 grains without apparently influencing the frequency of the cardiac irregularity.

These patients sought medical relief for the irregular action of the heart. The condition had recurred daily for months. They were afraid that they had serious cardiac disease. They had become very nervous and had difficulty in sleeping. They had lost their appetite and felt weak. They had lost from 5 to 15 pounds in weight; 3 had stopped their work because of weakness and others were contemplating this move. They had tried various remedies without relief. In 3 the premature beats were eliminated as far as the patients were concerned by the quinidin sulphate. In one of these an occasional skip was at times felt in the radial pulse, but the subject was unconscious of the irregularity. In the fourth the medication was apparently effective for about six weeks, and later seemingly had no influence on the prevalence of the premature contraction.

In the fifth the frequency of the premature beats was not diminished, even though the quinidin was increased to 6 grains three times a day. The first 3 were greatly benefited by the treatment; 2 have resumed their work and are apparently getting along well. The other one has recently reported that she feels better than she has in months. In each of these 3 the quinidin has been reduced to one dose a day. In 2 the capsule was taken after the evening meal and the other about one hour before bedtime. It was at these times that these patients were more prone to have skip beats. In each the one dose was at one time discontinued and the trouble returned. It may be added that these patients have been constantly encouraged. They have been repeatedly told that they have no serious cardiac trouble and to learn to disregard the premature beats if they happen to appear. The habits have been regulated as far as possible and gastro-intestinal disorders corrected. They were given occasional doses of bromid early in the treatment to assist them in sleeping. These measures have perhaps been partially responsible for the general improvement and may possibly have aided in the elimination of the skipped beats. In each instance, however, the premature contraction returned when the quinidin was discontinued. With the recurrence of the disorder the nervousness and insomnia returned.

Quinidin sulphate has in our experience eliminated or diminished the frequency of the premature contraction in 17 of 20 patients. In the 10 in whom the disorder was eliminated the medication has had to be continued once a day in 8. It would thus seem that quinidin deserves a trial in those in whom other measures fail. Even though the trouble may return after the drug is discontinued, these patients will be given an opportunity to recover from their highly nervous condition. There is apparently no objection to the use of quinidin in small doses over a long period of time should it seem advisable. Some of our patients have taken one capsule of 3 grains once a day for about four months without any ill effects.

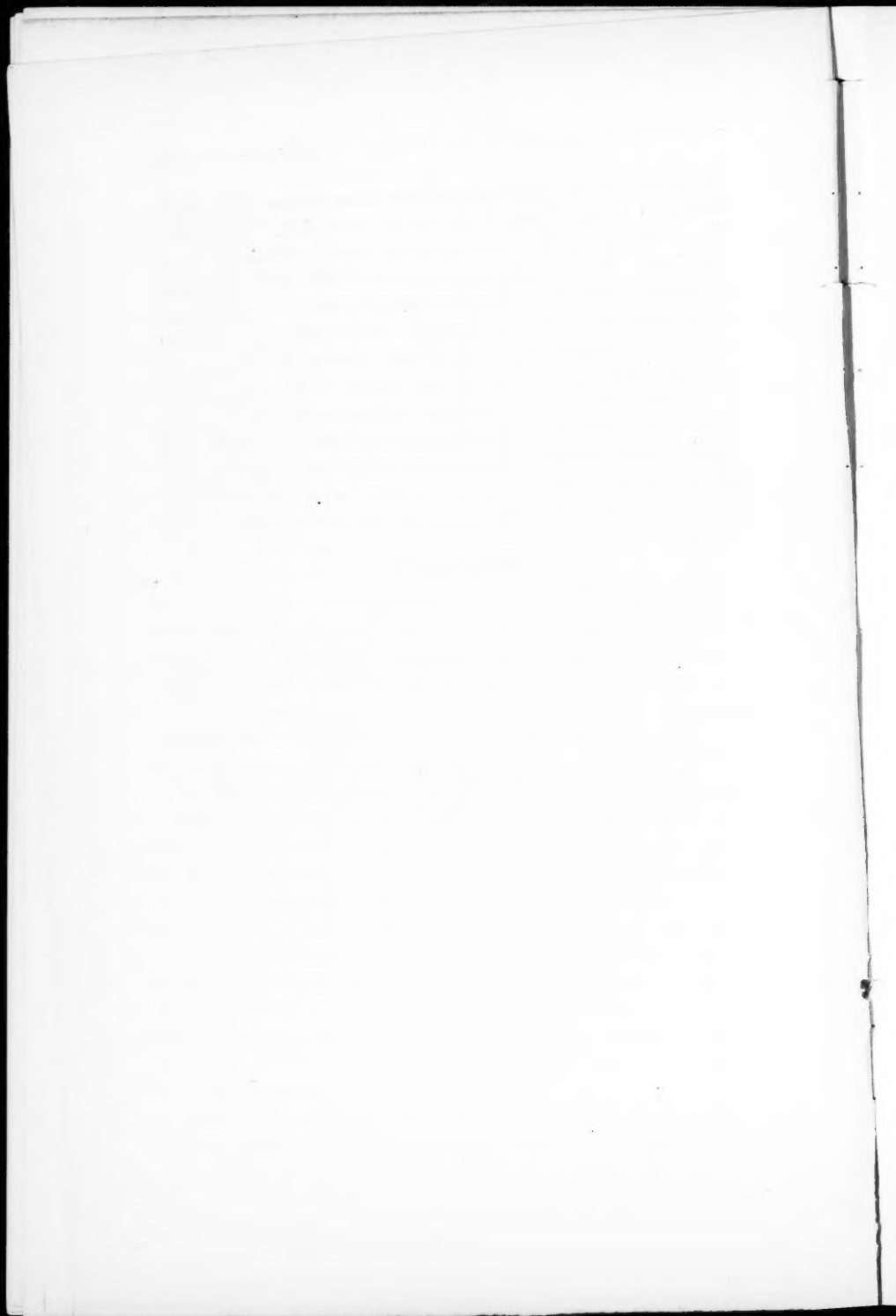
According to Lewis,<sup>5</sup> the most striking action of quinidin is the prolongation of the refractory period of the heart. He



has attributed the restoration of the sinus rhythm in auricular fibrillation to this change in the refractory period. The elimination of the premature contraction is perhaps explained on the same basis. In electrocardiograms of individuals with ventricular premature contraction show that the irregularity invariably falls at the same time in the cardiac cycle. The ventricular type often appears shortly after the T deflection. Lewis<sup>6</sup> has pointed out that the interval following the end of the refractory period of the normal cycle may be no more than a twentieth or even a fiftieth of a second. The stimulus that produces these contractions must, therefore, appear even earlier in the postrefractory period. It would then seem that a slight increase in the refractory period would interfere with the heart responding to the ectopic stimulus.

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## CLINIC OF DR. EDWARD LYMAN CORNELL

CHICAGO LYING-IN HOSPITAL

### NEPHRITIS IN PREGNANCY

**Patient Presenting Symptoms of Nephritis During Pregnancy. Treatment Before Labor. Craniotomy. Discussion of Nephritis in Pregnancy.**

THIS patient, Mrs. K., No. 19,904, presented for first examination June 2, 1921. The history at this time was as follows:

She was thirty years of age, para I, Jewish religion, born in Chicago. Her mother was living and well except that she had a high blood-pressure, averaging well over 200. Three brothers and 2 sisters were living and well. The patient had chickenpox and measles when a child. She had two abscessed teeth pulled within two years. She had a curetage for sterility about a year previous to examination.

Menstruation began at fourteen at intervals of twenty-five days. She flowed three or four days, a moderate amount with no pain. She had had no abortions. The last menstrual period was March 16, 1921. She had occasional attacks of vomiting and occasional headaches, but no constipation.

General examination revealed nothing unusual as far as lungs, heart, teeth, throat, extremities, and abdomen were concerned. A Wassermann test taken at this time proved to be negative.

The pelvic measurements were: interspinous, 26; intercristous, 28; bitrochanteric, 33; Baudelocque, 20; diagonal conjugate, 11.

The blood-pressure at this time was 143/90/80. The patient stated that she had frequent blood-pressures taken during the last three years, and at no time was it below 140. She had been

under treatment for increased blood-pressure. Her physician said that he had never found anything pathologic in the urine. The blood-pressure in this case, taken at bimonthly intervals, ranged between 140 and 150 until September 1st, when the patient suddenly developed severe headache, edema of the ex-

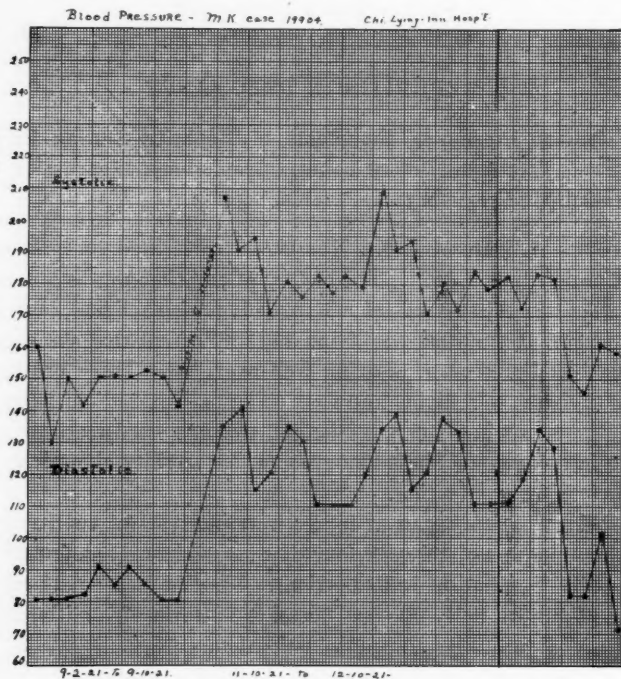


Fig. 310.—Blood-pressure readings charted from September 2d to 10th and November 10th to December 10th, the period of patient's stay in the hospital.

tremities, and numbness in the hands. She was sent to the Chicago Lying-in Hospital, and remained in bed on a non-protein diet and catharsis until September 10th. During this period her blood-pressure ranged between 142 and 160, as shown in Fig. 310. The urinalyses made daily did not reveal any albumin or casts. The metabolism tests, taken September 1st,

registered +12. The pulse during this time did not vary nor did she have a temperature. The patient was allowed to go home September 10th, with instructions to remain in bed the greater part of the day and to remain on a non-protein diet.

Between September 10th and November 10th, the date of her second admission to the hospital, the blood-pressure was taken at frequent intervals. It gradually increased in tension in spite of the diet and in spite of catharsis. On November 8th it registered 204/160/140. At this time severe headache was noted. The urine for the first time showed albumin and a few granular casts. It was with difficulty that the patient was persuaded to enter the hospital. Two days previous to her entrance the metabolism test showed +18, pulse was 92, and blood was taken for a blood chemistry test.

The phenolsulphonephthalein test showed 5 per cent. for the first hour and 30 per cent. for the second hour. The blood chemistry report was as follows: Non-protein nitrogen 46.1 mg. per 100 c. c.; urea 21.4 mg. per 100 c. c. This shows a retention and seems to bear out the nephritic nature of the toxemia.

When the patient entered the hospital on November 10th her blood-pressure was 208/135 at noon and 208/135 at 4.45 P. M. In the interval she had a hot pack and had sweated considerably. During the first twenty-four hours she was put on a starvation diet and given quantities of water. During this time she had considerable headache. On the 11th, following a hot pack, the headache was considerably better. It was my intention to induce labor at this time in spite of the fact that the patient was not due until December 23d. I hesitated, however, because the size of the uterus was such as to lead one to believe that she was not more than seven months pregnant. The uterus only reached 3 cm. above the umbilicus. I decided to call in Dr. R. T. Woodyatt, the internist. His report on the examination is as follows:

"The patient does not show any more distress than any active business man might. She does not give a history of headache, nausea, vomiting, or abnormal digestion or distress. The pupils react to light and accommodation and are equal. There is no nystagmus, exophthalmos, etc. The lungs and heart are

negative. The extremities show no edema. The reflexes are all normal except probably the knee reflexes, which are slightly exaggerated.

"The family history on her mother's side is not very promising as far as blood-pressure is concerned. On the father's side blood-pressure is negative as far as can be ascertained. The prognosis, as far as laboratory and physical findings show, offers nothing exceedingly alarming, and it would be quite justifiable to wait until more pronounced symptoms appear before interference.

**"Treatment.**—I would not recommend bleeding, from past experiences, which have been with somewhat different cases than the present. In the way of diet she should have 0.6 to 1 gram of protein to each calorie of body weight per day, with a sufficient amount of vegetables containing fluid and not containing more than 5 per cent. starch to keep up good elimination, with enough carbohydrates to prevent acidosis. She should be given enough food to make up 1600 calories per day. I would recommend alkalies sufficient to keep the urine neutral to litmus, but no more; sufficient water so the patient does not complain of thirst, and to keep the temperature below 99° F.; to get a good volume of urine hot packs should be given."

The patient was put on this régime, and the following notes were made of the development of the case:

November 17th: Patient apparently doing nicely; has not developed any eclamptic symptoms.

November 20th: Outlook very favorable.

November 24th: Patient's condition improving. Fetal heart tones were well heard throughout her period in the hospital.

November 26th: Patient in good condition. Fetal heart tones not heard.

November 27th: Fetal heart tones not heard; confirmed by three members of hospital staff.

November 28th: Patient's condition is good, but blood-pressure is increased.

At 4 o'clock on November 28th the patient complained of some abdominal cramps. The uterus was contracting on my

URINALYSIS

Name—M. K.

Date.	Twenty-four-hour amount.	Specific gravity.	Reaction.	Albumin.	Sugar.	Indican.	Urea.	Ammonia.	Acetone.	Diacetic acid.	Hyaline casts.	Granular casts.	Blood casts.	Leukocytic casts.	Blood.	Bacteria.	Pus.	Culture.	Remarks.	Examiner.
10/21	960	1024	Alk.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	65.43 solids	.....
10/16	960	1000	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	13.42 solids	.....
6/23	960	1002	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	13.42 solids	.....
6/30	960	1022	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	75.39 solids	.....
7/7	960	1028	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	39.03 solids	.....
7/14	A. M.	1026	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	epithelial cells	.....
7/21	1200	1014	Neut.	ft. tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	epithelial cells and 23.37 solids	.....
8/20	A. M.	1024	..	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	33.55 solids	.....
8/31	1440	1008	Alk.	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	33.55 solids	.....
9/1	A. M.	1020	..	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	33.55 solids	.....
9/29	960	1010	..	ft. tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	33.55 solids	.....
10/6	A. M.	1018	Neut.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1 hyaline	.....
10/18	A. M.	1014	Ac.	1 +	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	26.78 solids	.....
10/25	A. M.	1018	Alk.	3 +	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/1	A. M.	1016	Neut.	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/2	Single	1012	Alk.	2 +	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/8	960	1014	..	2 +	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/10	Single	1014	Ac.	4 +	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/12	..	..	..	hy. tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/14	..	..	..	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/15	Single	1008	Ac.	tr. +	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/17	..	1012	..	v. hy. tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/19	..	1015	..	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/21	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
11/23	..	1010	..	tr.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.....

Chart showing urinary findings from June 9 to November 23, 1921.

first visit at 9 o'clock the following morning. The uterus was longitudinal, the head in the inlet. Crepitus was felt. The breech was in the fundus and the back on the left. No heart tones were heard. At 11.20 A. M. rectal examination showed the bag of waters intact and the cervix effaced with 4 cm. dilatation. The pains were moderate at five-minute intervals. The patient was sent to the operating room and preparation made for delivery.

At 12.25 P. M. the patient is catheterized and given morphin, gr.  $\frac{1}{4}$ , with atropin, gr.  $\frac{1}{150}$ . It is decided to shorten the labor as much as possible because of the high blood-pressure. The patient is put to sleep, using ether anesthesia.

Vaginal examination showed that the cervix is 6 cm. dilated. Three small Dührssen incisions are made at 10, 2, and 6 o'clock. The cervix is cut a depth of  $\frac{3}{4}$  inch and a craniotomy done, using a Naegeli perforator, which is pushed through the skull at the anterior fontanel. The brain tissue is washed out thoroughly by means of a metal catheter, using a weak solution of iodine. The head is gently pulled through the cervix. The shoulders as well as the remainder of the fetus give no trouble in delivery. As soon as the fetus is removed we give  $\frac{1}{2}$  c. c. of pituitrin subcutaneously to cause contraction of the uterus. After waiting ten minutes the placenta is expressed. You will note that this placenta shows all the evidences of a nephritic placenta. It is small and the entire placenta does not cover an area greater than the size of my hand. On measurement we find that it is approximately 15 cm. long and wide. It is filled with red and white infarcts on the maternal side. There is one area the size of a half-dollar containing an old blood-clot.

The cervical incisions are now sutured with interrupted forty-day chromic catgut. The operation has taken thirty-five minutes. The perineum was not damaged. The patient left the table in good condition. The autopsy report (performed immediately) on the body of the child is as follows:

"The body is that of a fetus about 30 cm. long. The skin is macerated. There are several collections of fluids under the skin on the face. Most of the fetus was well developed, but small. The cord was of deep red color. On opening the abdominal



cavity a large amount of blood-stained fluid was found. The same type of fluid was found in the chest. The pericardium showed no important pathologic changes as the organs were removed intact."

At the time of delivery the patient's blood-pressure was 182/140/130. Immediately after delivery it was 150/95/80. By referring to the blood-pressure chart the variations occurring after delivery can be noted. The patient made a splendid recovery and left the hospital on December 10th. On only one day did she complain of headache. That was on December 2d. The uterus contracted nicely and no trouble was experienced with it.

This case presents many points of interest. The persistent high blood-pressure without corresponding findings in the urine is quite unusual. The absence of headaches and marked edema is also unusual in this type of case. The method of treatment taxes the judgment. It is difficult to predict what would have happened to the fetus if we had induced labor at the time the patient enter the hospital. The fetus weighed 1000 grams without the brain content on delivery. Would a fetus of this size have lived if born November 10th? The probabilities are that it would not.

During the past year I have had 4 cases which were quite similar. In 2 of these patients labor was induced, in one two weeks before the estimated term, and in the other four weeks before estimated term. In both instances the children lived. Each, however, weighed approximately 5 pounds. The one four weeks premature gave a little difficulty, necessitating incubation for about a week. The other gave no difficulty whatever. Both of these children are living at the present writing and doing nicely. The other patient was brought into the hospital two months before term and put on the same treatment as the present case. The fetus died *in utero* and was born small and macerated.

Whether we are to interfere or not interfere is the biggest problem that we have to meet. It seems to me that the following dictum may be made: Given a nephritic, high blood-pressure case with a fair size baby, with a mother not responding to treat-

ment, we had better interfere. Given a small size baby, such as occurred in this case, it is better not to interfere. In the latter case the child would be exceedingly difficult to raise because its resistance is so low. Also the odium which is attached to a case of this kind where the child dies after delivery is quite difficult to overcome.

While this patient had all the earmarks of a nephritic, it is probable that she would have developed eclampsia if the fetus had lived. My point in saying this is to call your attention to the difficulties often met in trying to state definitely that a case is certainly a nephritis pure and simple, or a true eclampsia with the kidney symptoms more pronounced. Cragin handles the subject of eclampsia and kidney of pregnancy under one heading, while DeLee attempts to separate them. Yet DeLee frequently says "see eclampsia." We often see patients who give all the signs and symptoms of acute nephritis in the latter weeks of pregnancy. Some of them develop true eclamptic convulsion and others none. In one case about a year ago, where the edema was truly terrific, the patient went into labor and delivered spontaneously. Within a few hours after delivery she went into coma, with periods of mild delirium. This state lasted about forty-eight hours. It was similar to the condition seen in uremic coma. She made a splendid recovery.

The treatment of these patients cannot be made routine. There must be a wide latitude. The general principles are practically the same throughout the country. In the diet we all attempt to eliminate proteins. At the Chicago Lying-in Hospital we have eliminated the milk diet in the past two or three years, substituting therefore a diet rich in carbohydrates. This régime was instituted after we had noted the rather frequent occurrence of convulsions postpartum in pre-eclamptics. Formerly all toxemias of pregnancy were put on a milk diet after delivery; now they are given a rich carbohydrate diet, which includes jellies, cereals, breads, starchy vegetables, citrous fruits, and plenty of water.

Elimination is promoted as far as possible by the kidney and intestinal tracts. Generally speaking, water is a good diuretic

and, unless the edema is too pronounced, water is pushed. Saline cathartics are used daily, enough being given to insure at least three watery movements a day. We use Epsom salts, milk of magnesia, and occasionally calomel and citrate of magnesia. Hot tubbing and hot packs are used rather sparingly except in the nephritic type of case.

In most of our patients the blood-pressure comes down if they are put to bed on the carbohydrate diet and given cathartics. In the exceptional case it is necessary to do a venesection. We do not like to do this previous to delivery for the reason one never knows how much blood a patient may lose after delivery, nor when immediate delivery may be necessary. We seldom use *veratrum viride* or nitroglycerin. Chloral hydrate is occasionally given per rectum if the patient is exceedingly restless. If the patient shows signs of impending convulsions, morphin sulphate, gr.  $\frac{1}{4}$ , every hour or so is given unless delivery is imminent.



## CLINIC OF DR. ELLIS KIRK KERR

COOK COUNTY HOSPITAL

### CASES OF VALVULAR HEART LESIONS

**Case I. Mitral Insufficiency and Stenosis in a Patient with Cardiophtosis, with Auricular Fibrillation, and Probably Renal Infarction.**

**Case II. Aortic and Mitral Insufficiency Due to Acute Endocarditis with Possible Aortic Stenosis.**

#### CASE I

THIS patient is of Scotch birth, forty years old, a plasterer by occupation: He entered the hospital March 3, 1922, with the following history: He had been home for a day or two on account of a cold, when he was suddenly seized with a pain in the right side of the abdomen radiating to the thigh. At the same time he was nauseated and vomited three or four times. He had no fever as far as he knows, but broke out into a cold sweat and became dyspneic. The pain lasted as above for a few hours, then moved to the upper abdomen, and became more of a dull ache. He was sent to the surgical service, with an examining room diagnosis of retrocecal appendicitis. The history which was written in the surgical service notes the irregular pulse and evident valve lesion. The temperature was normal, the leukocyte count 8600, and after a few days' study the patient was transferred to the medical service considerably improved. The medical history written by Dr. Richmond on March 7th repeats the above history of the present illness and notes that the patient has never had a similar attack previously. The patient has worked at his trade until the present illness began and has not had dyspnea, edema, or any other evidence of cardiac decompensation. He has had a cough for about five years. He gives

no history of any previous illness that we associate with acute endocarditis except that on direct questioning as to rheumatism he speaks of having some pain at times in the finger joints of the right hand. He had gonorrhea in 1911, but denies having had syphilis. He is married and has 5 children, all of whom are well. His father died at thirty-seven of unknown cause; his mother is well at sixty-four. He uses alcoholics and tobacco moderately.

Examination on admission notes that the patient is of medium height and rather spare build. He lay quietly in bed, but looked sick and perspired on very slight exertion. The pulse was irregular both in size of beats and spacing between beats—the *pulsus irregularis perpetuus* of the older writers. The temperature as recorded by the nurses was normal, respirations quiet and of normal rate. There was no cough present. Examination of the head and neck was negative. The chest is noted as being long and flat with an acute subcostal angle. It was symmetric and moved normally. Physical examination of the lungs was negative, the lower borders being at the usual height and showing a normal respiratory excursion. The heart apex was palpable in the fifth intercostal space in the mid-clavicular line, definitely outlined, wider than normal, heaving, and showed the same type of irregularity as the pulse. There was a peculiar flapping character to the apical impulse. A distinct presystolic thrill was felt at the apex. There were no other abnormalities on palpation. On percussion there was relative dullness in the second intercostal space on the left side as compared to the right, but definite dullness began at the third rib. The right border was 1 cm. to the right of the right border of the sternum. The left border on percussion coincided with the apex on palpation. On auscultation there was a blowing systolic murmur at the apex, and in the same region a rough presystolic murmur heard over a very limited area only. Both tones at the base were clear, but there was some accentuation of the second tone in the pulmonary area. The abdomen was normal in contour, but on palpation there was tenderness and an indefinite rigidity in the right upper quadrant. It was impossible to say whether or not the liver was enlarged; certainly

no definite border could be made out. The rest of the abdominal examination was negative. Rectal examination was negative. The genitalia were normal and there was no edema of the ankles. Examination of the urine was made daily and showed no abnormalities. The blood-pressure was 100 systolic and 75 diastolic. The Wassermann test of the blood was negative. Fluoroscopic examination of the chest showed normal lung findings and a moderate increase in size of both ventricles. Cystoscopic examination on March 14th by Dr. Culver showed a normal bladder. The urine from the right kidney showed a slight turbidity with decreased concentration suggesting a renal infarction.

On examination today (March 22d) the patient looks decidedly better. He has a better color, has lost the anxious expression he had, and does not sweat so profusely on any exertion. The apex-beat is smaller, less tumultuous, and is in the fourth intercostal space. The presystolic thrill has disappeared. The upper border on percussion is in the third intercostal space. The right border is still 1 cm. to the right of the right border of the sternum. The systolic murmur is still present, but the presystolic murmur is less in evidence. If, however, we listen carefully with the finger on the carotid, we note a murmur at the beginning of diastole, and reappearing just before the systole. This may be heard only in a limited area just inside the apex and you will find the murmur easier if you compare this area carefully with a point just to the left of the left border of the sternum at the same level. By shifting the stethoscope back and forth you can note the difference in the time of the sounds heard and can better analyze your findings. The abdominal symptoms and signs present on admission have disappeared.

The pulse shows the same type of irregularity present on admission. An electrocardiogram made yesterday shows auricular fibrillation, with one ectopic beat and slight left ventricular preponderance.

To summarize the clinical findings, we had a patient presenting himself with evidence of some acute abdominal condition and an evident valve lesion. From the clinical side the im-

portant thing to determine was the cause and nature of the abdominal trouble and whether or not operative interference was needed. Appendicitis, ruptured gastric ulcer, acute ileus, bile tract infection, and renal colic were suggested among other things, while some pulmonary lesion involving the diaphragm had to be ruled out. The latter was excluded by ordinary examination and by fluoroscopy. The absence of any rise of temperature and the normal leukocyte count made a diagnosis of any inflammatory process improbable, while the short duration of gastro-intestinal symptoms made ileus unlikely. The presence of a valve lesion suggested the possibility of infarction, but the location of the infarct remained in question until the ureters were catheterized and the urine from the right kidney found less concentrated than that from the left. It now seems probable that the patient had an infarct of the right kidney. The cause of the embolism must be left till the valve lesion has been analyzed.

The presence of a systolic murmur at the apex, enlarged heart, and accentuated second pulmonary tone shows the presence of mitral insufficiency, while the presystolic murmur on admission and the early and late diastolic murmurs present now justify us in making a diagnosis of mitral stenosis. The location and character of the apex are, however, peculiar for mitral lesions and suggest aortic insufficiency. The pulse is not that of an aortic leak and the second tones are clear throughout. The explanation of the unusual character of the apex appears on reviewing the data already given. It was noted above that the chest is long and narrow, a type in which a low position of the heart—cardiopsis—is frequently noted. As a rule the upper border in the ptosed heart is low—usually at the fourth rib. At present we find it in the third intercostal space, but on admission it extended an interspace higher. This would indicate that he had considerable dilatation of the left auricle on admission, which is, of course, in agreement with the presence of auricular fibrillation. The wide, heaving apex-beat noted above is due to the rotation that occurs in the ptosed heart which brings more of the left ventricle into contact with the chest wall. This



is a characteristic feature of cardiopaposis even when no lesion is present, and leads to considerable difficulty in diagnosis, when, as so frequently happens, there is a systolic murmur found. Then the presence or absence of an accentuated second pulmonary tone is of the greatest importance. At times a diagnosis may be impossible under these circumstances. In the patient before us the question of a possible functional murmur is easily settled by the presence of a diastolic murmur. It is a safe rule that a murmur occurring in diastole is never accidental, but always indicates some anatomic lesion. We will make an anatomic diagnosis, then, of a double mitral lesion occurring in a patient with cardiopaposis.

The etiology of the valve lesion and the cause of the embolism must next be considered. There are four groups of valvular lesions: first, those due to acute endocarditis; second, those due to a primary arteriosclerosis of the aorta involving the valve by exhaustion; third, the relative lesion where dilatation is so extreme that the opening is so large the valve cannot close it; fourth, congenital lesions. We have here no history of any of the usual diseases associated with acute endocarditis, but that happens often. The disease that is complicated by acute endocarditis may be so mild and the symptoms of the heart involvement so slight that no impression is made on the patient. The involvement of the mitral valve only and particularly the presence of mitral stenosis makes it very probable that this lesion is the result of acute endocarditis. It is possible to have a mitral valve involvement from primary aortic disease since the anterior cusp of the mitral valve arises from the aortic ring, but as a rule the aortic valve is also affected and stenosis under these circumstances is very unusual. Stenosis is, of course, impossible in the relative lesions. If then, we assume that the cause of the lesion was primarily acute endocarditis, the thought naturally arises that the present condition is due to an acute exacerbation of the old process with embolism due to a detached vegetation. The temperature has been normal throughout the patient's stay in the hospital, however, and the leukocyte count normal on two examinations. Then, too, the rapid improvement with rest

in bed speaks against an acute inflammatory process. The more probable explanation is that the embolus was due to a thrombus forming in the dilated auricle, passing to the left ventricle, and thence to the kidney. Embolism occurs most frequently in mitral stenosis of all the valve lesions because of the marked dilatation of the left auricle. Usually the emboli pass by the internal carotid to the brain, causing hemiplegia from obstruction of the middle cerebral artery. In this case, however, the thrombus entered the renal artery in all probability.

The degree of the lesion and the cardiac reserve must next be considered. The best measure of the degree of the lesion is the effect on the heart, and the location of the apex-beat is the best evidence of the size of the heart. At present the apex is in the fourth intercostal space and goes out about to the mid-clavicular line. The right border is 1 cm. to the right of the right border of the sternum. In other words, the heart is not much enlarged. The patient is perfectly comfortable, but he has been kept in bed since being in the hospital. When the patient first came under observation, however, the heart was much larger, the apex was lower, and the upper border higher. It was noted before that he had worked at his trade up till the onset of a "cold" a few days before coming to the hospital. He had never had dyspnea, edema, or other evidence of circulatory disturbance. It would seem that the present condition is about the same as before he was ill, that he developed an acute dilatation as a result of the "cold" he had contracted, and the renal infarction ensued. He has now, it would seem, recovered from the dilatation and is ready to resume a more active life. As to how rapidly he can return to his previous habits of life is a question. There have been various methods of estimating cardiac reserve proposed. Any method which may lead the patient to go beyond his limit of reserve may be harmful and should not be used. The patient should be tried out gradually on increasing amounts of exercise, being careful not to get dyspneic. As a rule we get our patients up as they improve, let them do more and more work about the ward till they are ready to go out. Their subsequent course is as much of a social as a medical problem, and

they are followed up by the social service department after leaving the hospital.

The prognosis in this case is then reasonably favorable in that the patient has reacted so favorably to treatment. He has evidently a heart muscle not much more impaired than would be normal for his age. It must be remembered that he has had acute dilatation once and may have it again. He has also mitral stenosis, which is apt to be progressive, and the auricular fibrillation has persisted so far.

The treatment of the patient has already been discussed. In the hospital he has had rest in bed and digitalis. The persisting auricular fibrillation raised the question of treatment for that condition. Favorable results are being reported from quinidin, but our observations are not yet well digested, and there is some likelihood of doing harm. This patient has no subjective symptoms due to the arrhythmia, and it would seem best to wait to see if the normal rhythm does not return. It seems to me that we should be very cautious in using a measure about whose mode of action we know so little, and when the treatment is used the patient should be watched very carefully and be frequently checked by the electrocardiograph.

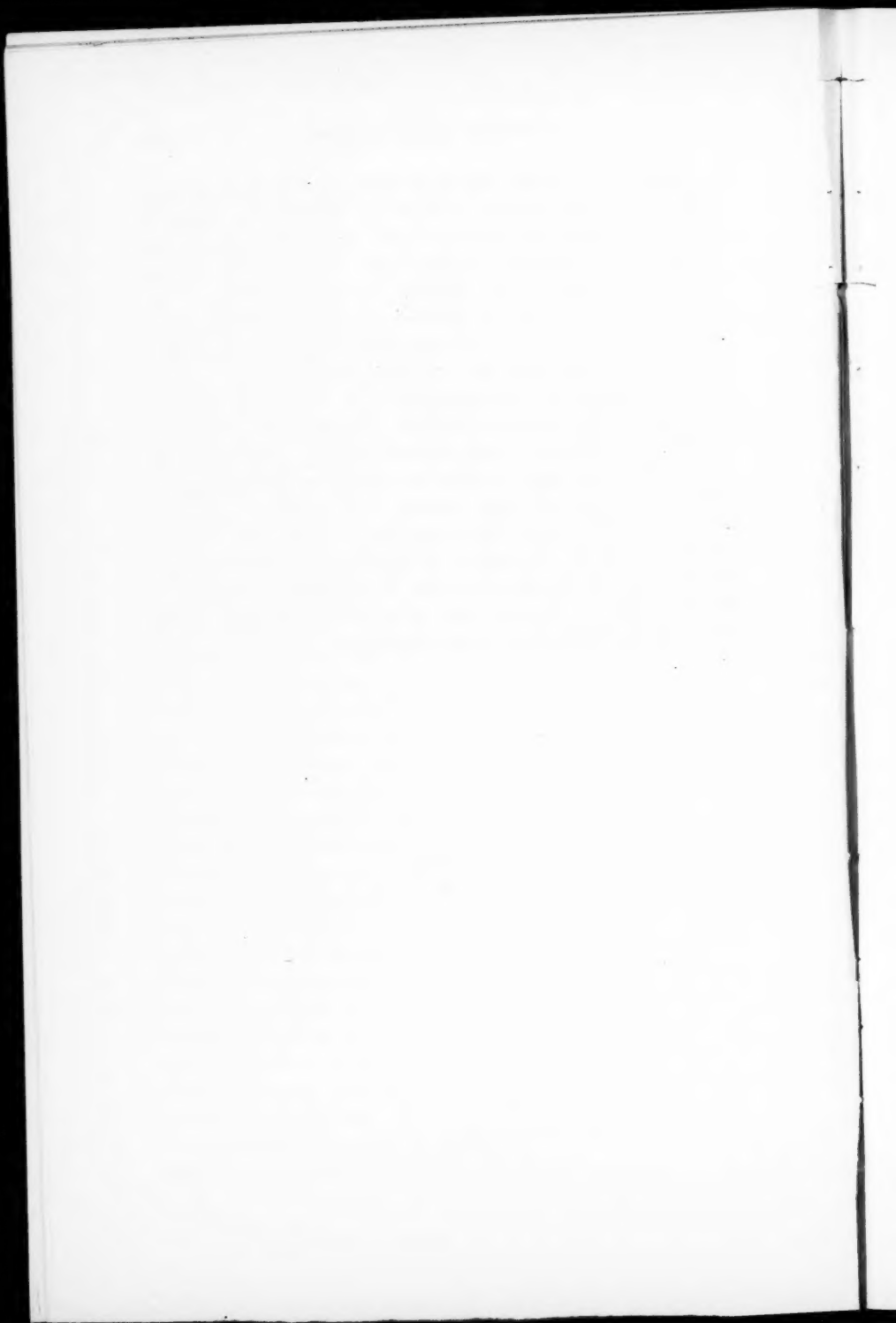
To summarize: We have a patient with a mitral insufficiency and stenosis with associated cardiophtosis. He was well compensated till he "caught cold," when acute dilatation and a renal infarction occurred. He has improved considerably and should soon be able to return to his ordinary duties.

#### CASE II

In the few remaining minutes I would like to sketch briefly another case and consider an interesting question in diagnosis. This patient is thirty-five years old and has a history of acute articular rheumatism. He has a valve lesion which has caused him no trouble, his present symptoms being largely those of a neurosis. Examination of the heart shows the apex displaced downward and outward, sharply localized, heaving, and wider than usual. He has a distinct systolic thrill in the aortic area. Percussion shows some enlargement to the left, but more to the

right. On auscultation there is a systolic and presystolic murmur at the apex, a distinct soft diastolic murmur heard best in the third left interspace, and a loud harsh systolic murmur replacing the first tone in the second interspace to the right of the sternum. The second pulmonary tone is accentuated. There is evidently aortic and mitral insufficiency. Whether the presystolic murmur at the apex indicates mitral stenosis or is the so-called Austin Flint murmur would seem to me to be impossible to determine and perhaps of little real importance, since we have no evidence of any considerable degree of stenosis. The interesting thing is whether or not to diagnose an aortic stenosis on the evidence at hand. The loud systolic murmur and thrill in the aortic area suggests the possibility of aortic stenosis, yet we know perfectly well that these signs occur very frequently without a stenosis. In fact, the usual teaching is that aortic stenosis is an extremely rare condition. The condition described in the books is very rare, namely, a hard, thickened, often calcified aortic ring with thickened fixed valve segments, all being the results of an arteriosclerotic process. There is another type, however, which is not infrequently seen at autopsy, the result of acute endocarditis, where the valve segments are adherent and thereby constrict the orifice. If these cases are seen at autopsy they should be seen clinically and in just this type of patient. Osler says never to diagnose aortic stenosis unless there is, in addition to the systolic murmur and thrill in the aortic area, a weak or absent first tone in the same place, and the characteristic pulse with its slow rise, sustained crest, and slow fall. In this patient the first tone in the aortic area is displaced by the murmur, and the pulse is not at all the typical water-hammer pulse of aortic insufficiency. It has a relatively slower rise, a more sustained crest, and a slower fall. The patient is young, has soft arteries, and the heart action is powerful, that is, all of the factors needed to produce a water-hammer pulse are present. It is usual to find a water-hammer pulse where both aortic and mitral valves are involved in an acute endocarditic process. It may be lacking where there is much peripheral arteriosclerosis.

There is a tendency among students and interns to make a diagnosis of aortic stenosis in rather an off-hand way whenever a systolic murmur and thrill is found. It has been the custom among many teachers to emphasize the extreme rarity of this lesion and to dispute this diagnosis on general principles. In his analysis of mistakes in diagnosis Cabot showed that errors of omission and of commission were most frequent in this lesion. There is no doubt, however, that this type of lesion, *i. e.*, one due to adhesions between segments of the valve, the result of acute endocarditis, is rather frequent. The presence of a definite combination suggesting aortic stenosis, namely, absent systolic tone in the aortic area, modified water-hammer pulse, together with the murmur and thrill, justifies us in making a tentative diagnosis of this lesion. Whenever any of these cases come to autopsy attention will thereby be directed to this possibility, and we hope in time to check our clinical findings. If this is not the proper conclusion, some other reason will have to be found for the modification of the pulse noted.



## CLINIC OF DR. JESSE R. GERSTLEY

MICHAEL REESE HOSPITAL

### FEEDING THE BABY, HIMSELF

The Essential of Infant Feeding is to Place the Welfare of the Entire Child Above That of Any Particular Group of Organs. If Clinical Judgment Decides that a Child with Diarrhea Needs More Carbohydrate, Then the Problem of the Pediatricist is Not to Treat the Diarrhea by Removing the Carbohydrate, but to Give the Carbohydrate in a Way Not to Increase the Diarrhea. If the Baby Vomits the Food which His Body Requires, the Problem is Not to Treat the Vomiting by Restricting this Food, but to Give it in a Way to Avoid Vomiting. The Index as to Whether the Local Symptoms are Affecting the Body as a Whole is the Body Weight. If the Weight Curve Falls, Then the Local Symptoms are Severe Enough to Require Treatment; if it Rises, the Local Symptoms May Be Disregarded. In the Latter Case Proportionate to the Gain in Weight and Improvement in General Nutrition is the Disappearance of Local Symptomatology. Perhaps the Most Important Effect of Attempting to Nourish the Body Cells Properly is to Give Them Strength to Withstand the Ever-recurring Parenteral Infections with the Associated Diarrheas.

SOME months ago 2 cases were shown illustrating what one might style "The Unemphasized Essential in Infant Feeding."<sup>1</sup> The point made was that pediatricists in seeking an easily digested infant food might at times overlook important essentials of general physiology. Food in the gastro-intestinal tract is essentially outside of the body. The function of digestion is to prepare this food for consumption. Once through the gastro-

<sup>1</sup> New York Med. Jour., August 3, 1921.

intestinal mucosa into the body itself food constituents offer material for growth and repair and supply energy for the daily work of the body cells. One eats in answer to these *body* requirements, and digestion simply changes food to a state pleasing to the body cells.

During the last year increasing interest in pediatrics has shown many disturbed nutritional conditions to be associated with vomiting and diarrhea. Frequent spurring, green, watery stools were symptoms sufficiently dramatic to sway entirely ideas of therapy. The fact that many of the worst cases of malnutrition did not vomit or showed normal or constipated bowel movements entirely escaped attention. The gastro-intestinal tract became a subject of overwhelming import and the fundamental purpose of feeding became subservient to the search for a food that could be easily digested.

These studies were not without value and did much to clarify our knowledge of infantile digestion. Often, however, the baby himself was sacrificed to the gastro-intestinal tract. The needs of the body cells were unrecognized.

A different philosophy, perhaps somewhat more radical, often gives happier results. Suppose one makes the fundamental object of feeding the effort to answer *body* requirements. Suppose one ignores local symptoms of indigestion unless they are causing damage *to the body*. The index as to this is the *weight curve*. If it drops, indigestion is sufficient to affect the baby's body, must be recognized, and treated. If it rises, however, abnormal gastro-intestinal activity is not sufficient to injure the patient. One simply continues the formula and general nutrition improves proportionately to the weight. Associated with the gain comes improved functioning of the gastro-intestinal tract. After a time gastro-intestinal efficiency becomes normal and local symptoms disappear.

The weight curves of two children both of the same age, both recovering from severe nutritional disturbance, illustrated this point. Each child had received tea and increasing quantities of albumin-milk for a period of three weeks.

Clinical observation has suggested that many children who



are recovering from these disturbances require increased carbohydrates to nourish their body cells. To give high sugar percentages in ordinary milk mixtures may lead to fatal diarrhea, but albumin-milk has solved the problem (Fig. 311, I). At A, the feedings have been increased to albumin-milk, 5 per cent. dextrimaltose, daily total 32 ounces. At B, dextrimaltose was increased to 7 per cent. At C, dextrimaltose was made 9 per cent.

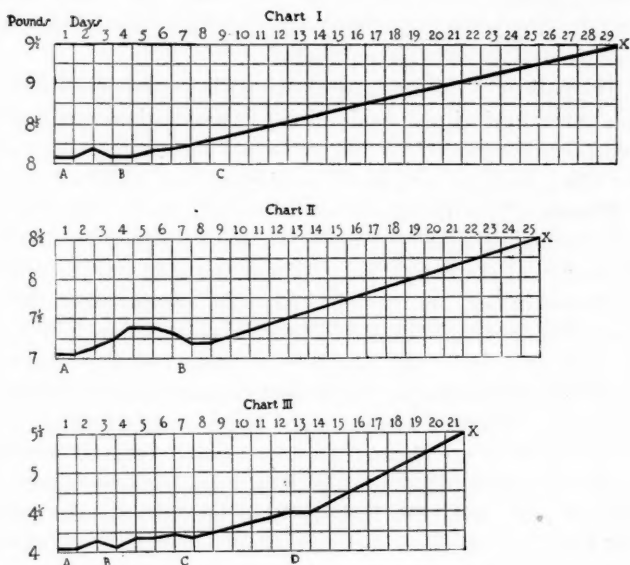


Fig. 311.—Charts showing weight curves in cases referred to in the text.

In this case, as the stools were normal throughout, there could be little question of the advisability of the increase.

In Fig. 311, II, the problem is more perplexing. Here gastrointestinal symptoms were overlooked entirely, to meet the greater demands of the child's body. At A the diet was albumin-milk with 5 per cent. dextrimaltose, 31 1/2 ounces daily. At B one faced a grave problem. The daily six soft, green stools suggested an increasing intolerance of the intestine. The dropping weight

curve showed the need of the body for food. Should one treat the intestine and withdraw the food? Should one treat the body and increase? With a child on albumin-milk under close observation and in a fairly satisfactory condition it is permissible to follow the latter procedure. At B dextrimaltose was increased to 7 per cent. The immediate response of the child's general condition led to a proportionate increase in the efficiency of the gastro-intestinal tract and within two weeks the stools were normal. It must be emphasized, however, that this technic can be employed only with albumin-milk.

The patient of this morning illustrates in another fashion the same general principle. Again local gastro-intestinal symptoms have been disregarded for the welfare of the entire child, and once this latter is accomplished, local symptoms subside.

**History.**—This infant is eleven weeks old. At the age of five weeks he was brought by a caretaker who knew nothing of the first month of the baby's life. At four weeks of age the child was placed in her care. For five days she offered, every three hours, a bottle of half-milk and half-water. He took only from 1 to 1½ ounces at a time. The bowels were regular and of good quality until the last two days. Stools then became frequent, watery, and green. Vomiting was acute and loss of weight striking.

At that time examination showed a puny, cyanotic infant. Weight was approximately 4 pounds. The sunken fontanel, hollowed eyes, wrinkled, inelastic skin proved a severe malnutrition. The diarrhea and beginning fever left him almost moribund from a complicating alimentary intoxication.

Under the usual emergency measures, 60 c. c. of saline intraperitoneally, camphor and oil hypodermically, and very small quantities of breast milk, life was spared. The breast milk was increased gradually until he got eight feedings of 2½ ounces (Fig. 311, III). For a period of about one week ending just after "A" he took this well, but the stationary weight showed a failure to gain. At B breast milk was increased to eight feedings of 3 ounces. This was the limit as to quantity. The child would take no more. What was to be done? This period of failure to gain following acute nutritional disturbance has long been noted and

considered normal or even excusable as "the reparation period." However, a bit of physiologic reasoning may enable us almost at will to dispense with it. What considerations lead to proper therapy?

Here is a child getting breast milk, getting as much as he will take and not gaining. In the two previous cases on albumin-milk we could increase the carbohydrate. It does not seem feasible to attempt this with breast milk. Is breast milk the ideal food for a baby of this type? For a normal, sound infant no food is better, but breast milk is not rich in protein or mineral matter, the very essentials of body structure and probably the constituents in which the patient is deficient. Maybe the addition of a little food easily digested but containing more of these ingredients would be of value. Buttermilk, for instance, has all the protein and mineral matter of cow's milk, but has no fat or carbohydrate. To replace the loss of fuel value and to supply carbohydrate which is so essential to these children one could prescribe a buttermilk mixture.<sup>1</sup>

This mixture offers good fuel value, ease of digestion, and more protein and mineral than is contained in breast milk. In a way the principle is much the same as the albumin-milk and high carbohydrate feeding of the previous curves. The lactic acid of the buttermilk has no significance in this scheme. One starts by adding a small quantity of the mixture to the breast milk and then increases as indicated. It is not wise, however, to give in a day a quantity more than one-half the total of breast milk. At C the following mixture was prescribed: Breast milk 2½ ounces, buttermilk mixture ¼ ounce, eight feedings of 3 ounces. The immediate response of the weight curve showed that at last the demands of the baby's body were being answered. At this stage the child for the first time started to vomit from one to four times daily. However, one follows the same fundamental philosophy of the first 2 cases. As long as the weight curve was rising local symptoms could be disregarded. At D for a few

<sup>1</sup> To 32 ounces of buttermilk one adds 2 ounces of cane-sugar and 2 ounces of wheat flour. Stir this until it is smooth, and then boil twenty minutes, stirring constantly.

days the weight curve remained stationary and feedings were changed to breast milk 2 ounces, buttermilk mixture 1 ounce, eight feedings of 3 ounces. Again the curve began to rise, and with the associated improvement of the baby's general nutrition gastro-intestinal activity became normal and vomiting ceased. One cannot emphasize often enough that the best index of a baby's nutrition is the *weight curve*, and when this rises all local gastro-intestinal symptoms may be disregarded. If these symptoms are severe enough to require treatment, loss of weight gives immediate warning.

In discussing the fundamental importance of the body tissues themselves a recent study from the clinic of Dr. L. F. Meyer is of interest. It has long been known that state of nutrition and infection are closely related. Indeed, this might be described as a reversible reaction.

Infection:—————Nutrition

Well children suffer infections followed by secondary disturbances of nutrition. Children with disturbances of nutrition are especially prone to infections. By infection is meant such "grippal" complications seen during the winter months as coryza, nasopharyngitis, otitis, and bronchitis.

Two of Meyer's assistants, Wertheimer and Wolff, studied the question from a statistical point of view. In large groups of infants they noted the relation of age, diet, and state of nutrition to the frequency, course, and prognosis of various infections.

The number of infections varies whether a child has individual or institutional care. In the home an infant goes through approximately three infections a year. In an institution the number is nearly four during the first six months.

The *frequency* of infections depends not on diet, but on *age*. Children on breast milk or on cow's milk were affected alike. On an average those below three months of age suffered an infection every two months; those of five to six months, every month. Tabulating the average number of infections in their institution, they found:

	Per month.
Breast-fed child.....	0.52
Healthy bottle baby.....	0.59
Premature.....	0.56
Non-thriving child.....	0.61
Malnutrition or decomposition.....	0.56

So diet does not affect the frequency. Note, however, the effect of age:

	Number of infections decidedly increased at	
Breast-fed baby.....	6 months	
Healthy bottle baby.....	6 months	
Non-thriving baby.....	4 months	{ Get as many as the healthy child at six months.
Premature and weakling.....	3 months	
Malnutrition or decomposition.....	3 months	

So the *number* of infections does not depend upon state of nutrition, but upon the age.

Next let us note the duration:

	Lasted on an average
Breast-fed baby.....	0.5-1 week
Healthy bottle baby.....	2.5 weeks
Non-thriving baby.....	3.8 "
Decomposition and premature.....	4.5 "

Here for the first time does one see the influence of diet and state of nutrition.

Lastly note the prognosis:

	Mortality, per cent.
Breast-fed baby.....	0
Normal bottle baby.....	10-28
Prematures and non-thriving babies.....	40
Decomposition.....	90

These studies reveal the important relation of state of nutrition to resistance and immunity. That they are so striking is probably due to the long period of institutional life. All individuals are born apparently with a resistance of varying duration. In the premature this is short. At three months, when his endogenous supply of iron is failing and when rachitic changes and increased electric irritability suggest changes in calcium metabolism, the number of infections rises to equal that of the

breast and healthy bottle babies at six months. So it is with the baby suffering with malnutrition. Is this also an argument for a general mixed diet at six months even in the breast fed? I have repeatedly emphasized the good clinical results following such a schedule.

While of no importance as regards inherited immunity, the diet and state of nutrition are of undeniable importance in influencing the severity, duration, and prognosis of infections.

Pediatricists for some years have leaned to the idea that diarrhea in artificially fed children is associated more frequently with these "grippes," these so-called parenteral infections, than with unmistakable errors in diet.

Perhaps after all the great difference in the effects of breast milk and cow's milk mixtures is that in the former the combination of food elements is such as to give perfect nutrition to the tissues. These then become resistant to many of the usual types of infection.

In artificial feeding our interest has centered so completely upon making the mixture acceptable to the gastro-intestinal tract that we have forgotten the fundamental demands of the body cells. Is it not possible that many diarrheas previously attributed to the indigestibility of cow's milk may be the intestinal reaction of a baby whose imperfectly nourished body cells are unable to offer proper resistance to infection? These are studies for the future. At any rate, in feeding formulæ, do not forget the baby.

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